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HISTOPATHOLOGIC OBSERVATIONS IN SO-CALLED CONGENITAL FIXATION OF THE STAPEDIAL FOOTPLATE.

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INTRODUCTION.

Studies during the past decade by Shambaugh[1,2] Holmgren,[3] House,[4] and Hoople[5] have led to the recognition of congenital footplate fixation as a clinical entity. Their studies have all been based on clinical examinations and observations at operation. The case reported in the present paper is the first to be studied histologically.

The patient with "Congenital Footplate Fixation" is born with a severe hearing impairment. If this condition is bilateral, as has been observed so far in the majority of instances, development of speech is retarded, and speech is

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much impaired. The clinical condition may be recognized at an early age. Cochlear function studies at a later age reveal a pure conductive type of loss, averaging approximately 50 db throughout the speech frequency range. Bone conduction is usually not impaired, and speech discrimination is normal, indicating an intact cochlear end-organ. Repeated hearing tests on these patients over years, as they have been done by Shambaugh[1,2] and House,[4] revealed that the amount of conductive hearing loss remains unchanged. The hearing impairment, therefore, is not progressive. A positive family history of this malformation, even if it is associated with developmental anomalies of the middle and external ear, is extremely rare (Altmann[6,7]). Congenital Footplate Fixation may occasionally be associated with congenital atresia of the contralateral ear or with a malformation of the lower jaw.

The surgical explorations that have been made reveal that the annular ligament is practically absent and that there is no evidence of an otosclerotic lesion involving the oval window. The osseous plate obliterating the vestibular fenestra may vary in thickness and appearance. A dehiscence in its central portion, covered by a thin transparent movable membrane, has been reported in one case by Shambaugh.[1]

The patient with "Acquired Footplate Ankylosis," as a result of otosclerosis, is born with normal hearing, and development of speech is not affected. Impairment of hearing begins insidiously, most often between puberty and the early thirties. One ear may be impaired before the other, but usually both ears are eventually affected. Cochlear function studies reveal that the hearing loss is conductive in nature. Hearing impairment is usually progressive, but profound deafness, to the extent that the use of amplification is not beneficial, is rare. In 50 to 60 per cent of instances, there is a positive family history of this disease.

Exploratory tympanotomy in most instances reveals a single circumscribed, clearly demarcated otosclerotic lesion between the anterior border of the oval window and the cochlea. Ankylosis occurs by otosclerotic bone growing across the annular ligament and involving or replacing the adjacent segment of

the footplate and the base of the anterior crus. In the areas where the footplate is not involved by the otosclerotic lesion, its margin and the annular ligament are distinct. The footplate there is thin, transparent, and shows a characteristic bluish-grayish shade. With the operating microscope, the appearance of otosclerotic footplate fixation is, therefore, quite different from that seen in congenital fixation.

EMBRYOLOGY.

The term "Congenital Footplate Fixation" appears not to be the appropriate one to characterize this malformation, since it may give the erroneous impression of an ankylosis having occurred at some developmental stage during embryonic life. Bast and Anson[8,9] in their exhaustive studies on the development and the anatomy of the ear and the temporal bone in man, have clearly shown that the stapes is derived from two sources, namely, the second visceral (hyoid) bar and the lateral wall of the otic capsule.

The origin in the visceral bar is primary and provides the greater mass of the stapes, the annulus stapedialis. Subsequent fusion with the lamina stapedialis of the otic capsule and secondary differentiation of laminar tissue provide the stapedial rim, the vestibular surface of the base and the basal perichondrium. Further modification of the peripheral laminar-tissue leads to the formation of the annular ligament around the stapes.

In the 50 mm. (10-11 weeks) embryo, the base of the stapes is separated from the marginal tissue of the vestibular window and through alteration of the precartilaginous tissue in what was, earlier, the lamina stapedialis. Since this zone, the site of the future annular ligament, is merely one in which chondral matrix is present in smaller amount than in either the adjacent tissue of the base or that of the fenestra, the stapes at this stage of development is histologically continuous with the vestibular "window" and, in the sense in which the term fenestra is used to describe the opening in the adult bone, no window exists in the otic capsule of the early fetus.

The extensive reports of Bast and Anson and their as-

sociates[8,9,10,11] give the details of the changes that result in the formation of a normal stapes, oval window and annular ligament. For the present paper, it suffices to point out that the maldevelopment resulting in congenital fixation must occur at stages later than the 50 mm. one.

CASE REPORT.

These temporal bones were obtained by Dr. Sam H. Sanders of Memphis, Tenn. They are from a 29-year-old white man with the following history: The patient's first hospital admission was at the age of seven for study after a fall, followed by a transient episode of unconsciousness. The records mention a recurrent chronic bilateral otitis media and almost complete deafness in both ears, but give no details. Congenital mental deficiency was the principal diagnosis.

The second admission was at the age of 18 to a State Hospital. The patient had wandered from home and, being unable to understand directions from the authorities, had had difficulties with the police. On examination he gave a past history of several episodes of pneumonia and purulent drainage from both ears until the age of 12. The physical examination revealed that he was small for his age and that his teeth were in poor condition. He was "deaf" and unable to speak intelligibly; however, he could read, write, and was a good lip reader. He was mentally deficient. Otherwise the physical examination was negative. The family history was non-contributory. The patient stayed in the hospital for seven years and worked well on several assigned duties. Since he had been doing well, he was discharged and employed with his father in a printing shop; but when after six months he wandered aimlessly away from home on several occasions and he was readmitted at the age of 25. A psychological examination during this period described the patient as mentally deficient and emotionally unstable. No mention of illness or of unusual physical change is found in the records until his terminal illness.

One day the patient was reported to have vomited, and the ward physician felt that he looked and acted ill, although he was afebrile, and the physical examination was negative. At 8:00 A.M. the following day, the patient appeared acutely ill; his neck was stiff and Lasègue's sign was positive. His throat was red; he was still afebrile. Antibiotic and sulfa therapy was started. Later in the day his temperature rose; he became quite restless and death occurred at 4:15 P.M.

The postmortem examination revealed pneumonia and an acute purulent leptomeningitis due to diplococci. Bronchiectases were found in the right middle lobe.

METHOD.

The temporal bones were removed four hours after death and received four months later in the University of Chicago Ear, Nose and Throat Laboratory in 10 per cent formalin. Decalcification was done with 5 per cent trichloracetic acid and embedding in celloidin. Both bones were sectioned in the

horizontal plane at 20 micra. Every fifth section was stained with hematoxylin and eosin for histologic studies.

HISTOPATHOLOGY.

The histological examination of both temporal bones revealed in summary:

1. Developmental anomalies of the labyrinthine capsules involving the internal auditory canal, cochlea, vestibule, cochlear and vestibular aqueducts and the vestibular fenestra.

2. Solid bony ankylosis of the stapedial footplates; minor structural deformities of the stapedial arches.

3. Extensive malformations of the membranous labyrinth and of the first neurons of the cochlear and vestibular nerves.

4. Multiple areas of bone resorption with partial replacement by granulation tissue throughout the labyrinthine capsule.

5. Numerous fistulous tracts, as a result of bone resorption, establishing open pathways between the internal auditory canal, cochlea, vestibule, semicircular canals and middle ear.

6. Leptomeningitis, purulent, acute. Labyrinthitis, purulent, acute, caused by extension along the cochlear and vestibular nerves, and by way of the numerous fistulae in the labyrinthine capsules. Osteitis and mastoiditis, purulent, acute.

The histopathologic findings to be reported in this communication will be limited to the area of the vestibular fenestra. A separate paper will be devoted to the description and interpretation of the developmental anomalies of the inner ear and to the character and distribution of the osseous lesions encountered in the otic capsule.

HISTOPATHOLOGICAL EXAMINATION OF THE OVAL WINDOW AREAS.

Right Ear.

There is an almost complete osseous union between the footplate and the adjacent portion of the otic capsule. This

synostosis is extensive all along the fenestral rim except for a small area at the posterior end of the window. In one section (see Fig. 1) the union is fibrous only. There, one encounters a very thin layer of cartilage on the stapedial rim and on the adjacent region of the vestibular fenestra. In other sections (see Fig. 2) the union is partly fibrous, partly cartilaginous and partly bony.

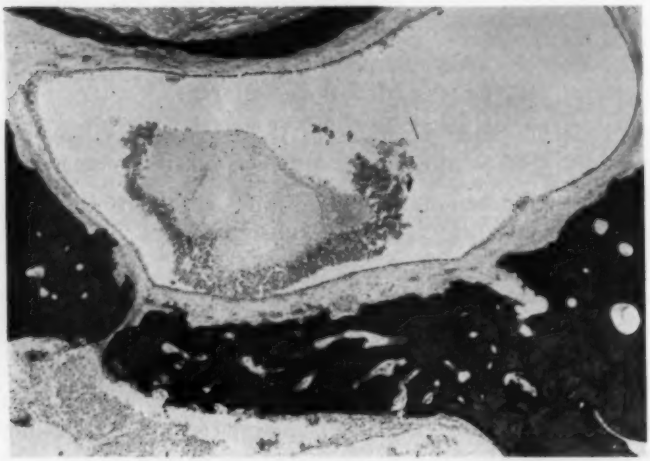

Fig. 1. Right ear. Section No. 280. X50. Superior portion of the footplate. Rudimentary annular ligament is seen at the posterior end only. Anteriorly, the footplate blends into the vestibular capsule.

In the region of the fibrous union is one area where the collagen fibers are dense and in parallel arrangement. This suggests the formation of an annular ligament (Rt. No. 280). There is no evidence of this ligament elsewhere. The dimensions of the footplate are normal except for its thickness. It is thicker in the marginal areas than in the center, but there is no dehiscence. Wherever the footplate blends into the vestibular capsule, the structure of the bone does not vary from that of the otic capsule. The bone reveals mainly a simple laminated structure, characteristic for endochondral bone, but there are areas in the posterior crus and in the otic

capsule where bone shows Haversian type of ossification (Rt. No. 300).

The inferior half of the footplate reveals a two-layered structure. The inner layer, next to the vestibule, is continuous with the labyrinthine capsule; the outer layer, next to the middle ear, is continuous with the crura of the stapes. The dividing zone between the two is formed by an inter-

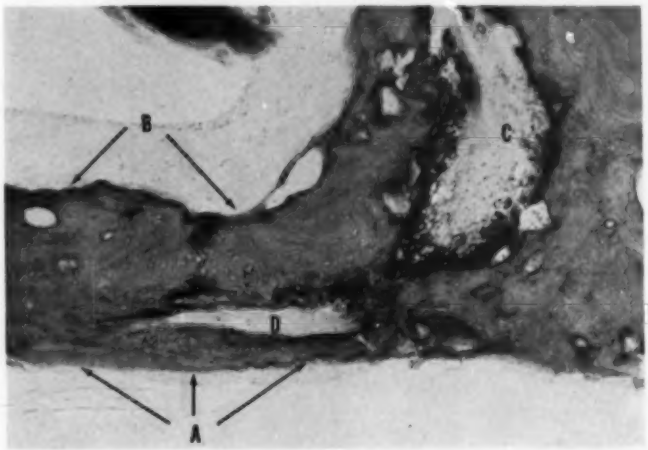

Fig. 2. Right ear. Section No. 310, X155. Posterior end of the footplate. The footplate appears as a two-layered structure, an inner layer (A) continuous with the labyrinthine capsule and an outer layer (B) continuous with the crura of the stapes. The dividing zone between the two is formed by an interrupted layer of cartilage (C) or fibrous tissue bordered by cartilage (D). This bilaminar structure strongly suggests an incomplete fusion of the annulus stapedialis with the lamina stapedialis and failure of the latter to separate from the otic capsule.

rupted layer of cartilage or fibrous tissue bordered by cartilage. This bi-laminar structure strongly suggests an incomplete fusion of the annulus stapedialis with the lamina stapedialis (Rt. No. 310), also a failure of the primitive stapedial lamina to separate from the otic capsule, as it normally does.

In the superior portion of the footplate, the inner lamina is mostly resorbed except for a small lip of osseous tissue pro-

truding from the anterior window margin on the vestibular side. The remaining footplate consists mainly of the tympanic layer, derived from the hyoid bar (see Fig. 3).

The crura are bent downward markedly, so that they join the base at an angle acute inferiorly. The crural arch is ovoid, and the crura are channeled on the obturator side. This hollowing extends into the capital end of the ossicle;

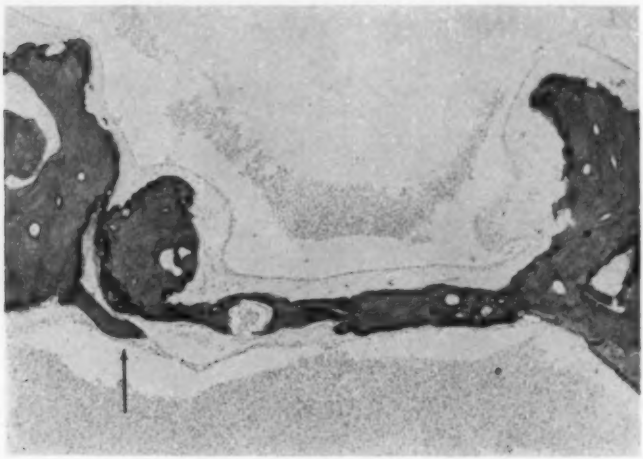

Fig. 3. Right ear. Section No. 300, X60. Note the small lip of osseous tissue (arrow) protruding from the anterior window margin on the vestibular side. The remaining footplate consists, mainly, of the tympanic lamella, most of the inner lamella having been resorbed.

neck and head show marked excavation. The incudostapedial articulation is essentially normal. Vascular channels are present in the footplate and in the base of each crus. There is no evidence of any stage of an otosclerotic process anywhere in this region.

Left Ear.

The histopathological findings are similar to those encountered on the opposite side except in one respect: the bony union is complete with the exception of a small area at the

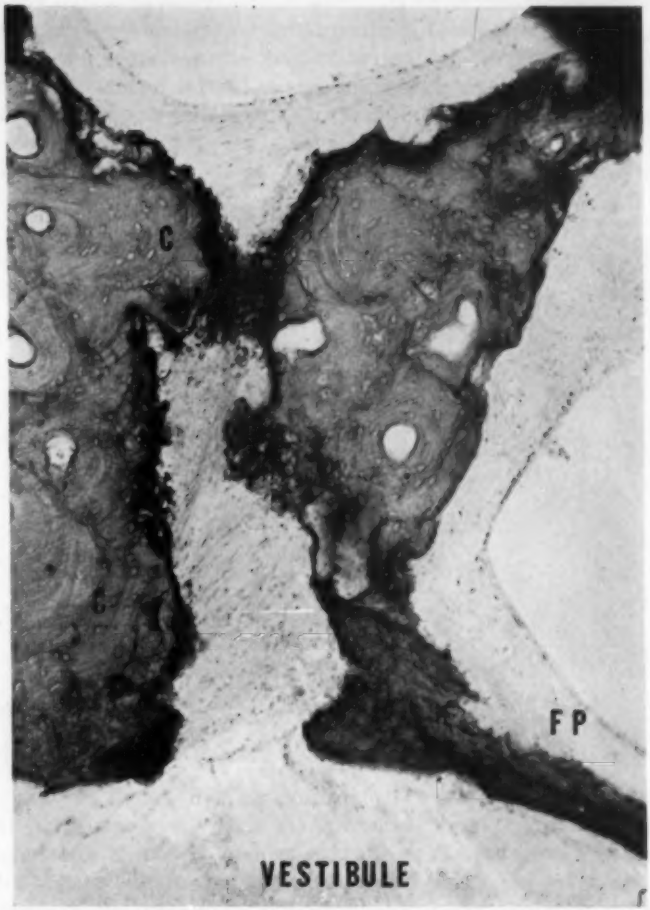

Fig. 4. Left ear. Section No. 320, X165. Showing the anterior margin of the footplate (F.P.) and the adjoining rim of the capsule (C). An annular ligament has been partially formed.

posterior margin of the fenestra. Where the union is fibrous, a calcified chondral plate forms the margin of the footplate as well as the adjoining rim of the vestibular fenestra. In that area also, the concentration and arrangement of the collagen fibers is such as to indicate the partial formation of an annular ligament (see Fig. 4). No cartilage is present on the vestibular side of the footplate, as on the opposite ear. Elsewhere the ligament is absent or incompletely formed.

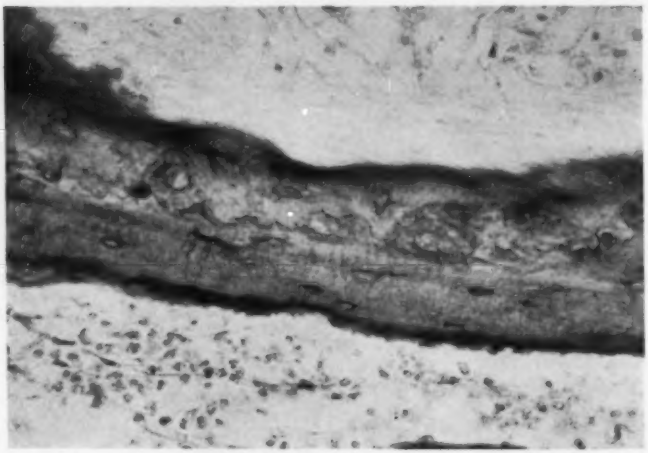

Fig. 5. Left ear. Section No. 301, X600. Midportion of the footplate showing a simple laminated structure with the occasional formation of Haversian systems.

The diameters of the footplate are normal, except its thickness, which is more marked in the marginal portion. The center is thin, but shows no dehiscence. The bony structure is the same as in the otic capsule. It is a simple laminated structure with occasional formation of Haversian systems (see Fig. 5).

A bi-laminar structure of the footplate is suggested in the inferior portion (see Fig. 6). A layer of fibrous tissue bordered by cartilage or of cartilage tissue alone forms the zone which separates the inner, vestibular, from the outer, tym-

panic lamella. In the superior portion of the footplate, the inner lamella is partially resorbed. Its remnants are indicated by lingular osseous protrusions from the anterior and posterior window margins on the vestibular side (see Fig. 7). As in the other ear, the osseous union between footplate and otic capsule is formed superiorly by the outer lamina; inferiorly, by the inner one.

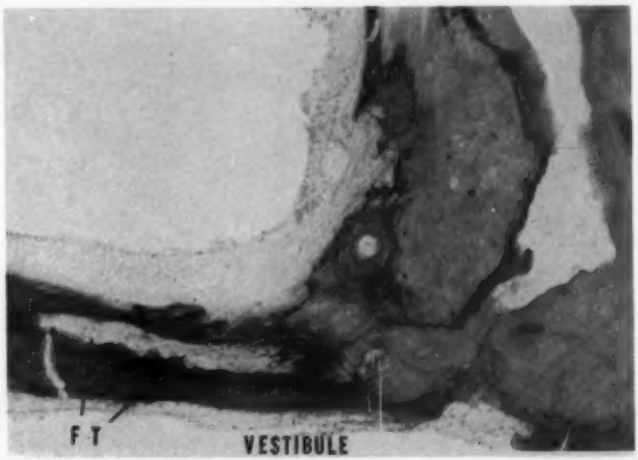

Fig. 6. Left ear. Section No. 236, X180. The junction of the posterior margin of the footplate and the otic capsule. A bilaminar structure of the footplate is suggested by the presence of a layer of fibrous tissue (F.T.) which apparently forms the zone which separates the inner vestibular from the outer tympanic lamella.

The crura reveal the same structure, configuration and anomalies as on the opposite side. Blood vessels in the bone are present in the bases of the crura and in the footplate. There is no evidence of otosclerotic bone in the area of the oval window or elsewhere in the otic capsule.

DISCUSSION.

The anatomical studies on these temporal bones are in agreement with the clinical observations made by Sham-

baugh,[1,2] Holmgren,[3] House,[4] and Hoople.[5] The vestibular fenestra may be obliterated by birth and by a process other than otosclerosis. In this respect, "Congenital Fixation of the Stapedial Footplate" does exist, and it may be considered a clinical entity.

Otosclerosis, Paget's disease, Endemic Goiter (iodine deficiency goiter), adhesive processes and forms of localized osteitis, etc., are among the pathological processes in the

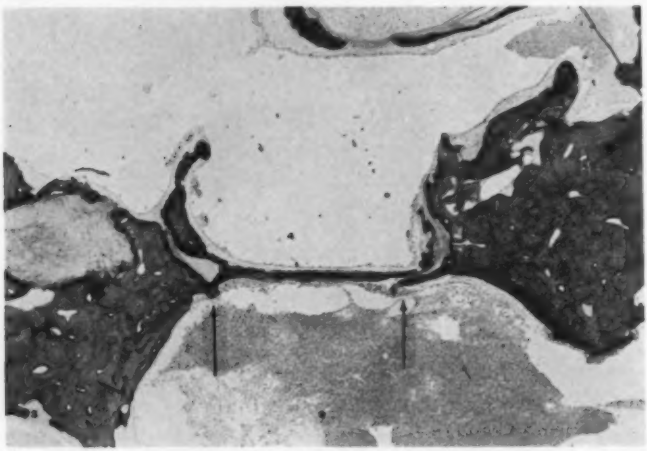

Fig. 7. Left ear, X28. The inner lamella is partially resorbed, leaving as remnants, lingular osseous protrusions (arrows) from the anterior and posterior margins.

middle ear or otic capsule, which may lead to new bone formation and thus to fixation of the stapedio-vestibular articulation. Perusal of our sections, however, excludes any of these morbid processes from having been the cause for a secondary ankylosis in a previously normally developed oval window; hence, the osseous closure must have a different etiology.

Developmental anatomy reveals that no "window" exists in the otic capsule of the early fetus in the sense in which the term fenestra is used to describe the opening in the fully

developed temporal bone. The annulus stapedialis, a derivative of the second visceral bar, fuses with the lamina stapedialis of the otic capsule to form the future stapes. As the base of the stapes increases in diameter, the cartilaginous fenestral rim undergoes dedifferentiation from cartilage to mesenchyme. The base of the stapes widens by a concurrent process of decrement on the capsular, and of increment on the stapedial side until the stapes has attained adult dimensions. This occurs at about mid-term stage. It is not until the fetus has reached this stage that an annular ligament becomes established. This ligament comes into being by a transformation process of the precartilaginous tissue in what was, earlier, the lamina stapedialis (Bast and Anson[8,9]).

The serial sections through the vestibular fenestrae clearly indicate a bi-laminar structure of the stapedial base. These two laminae are separated by a zone of fibrous tissue bordered by cartilage or of cartilage alone. At the posterior end of the footplates, where a fibrous union exists between the base and the window margin, a thin layer of cartilage exists on the circumference of the base and on the adjacent margin of the oval window; also, a parallel arrangement of collagen fibers is present in that area, but more definite in the left ear, and this could be interpreted as a partial formation of an annular ligament. The sections reveal further a structural deformity of the crural arches.

Obviously no osseous transformation process, whatever its etiology, can produce such pathological changes in a footplate. These observations, however, can be explained on the basis of an anomalous development of the stapedial footplate and the adjoining otic capsule.

Whereas investigators have been in general agreement on a branchial derivation for the malleus and incus, the opinions in the past have differed widely as to the origin of the stapes (Cauldwell and Anson[12]). The opinions were that the stapes arose from *1.* the first branchial (mandibular) arch, *2.* the second branchial (hyoid) arch, *3.* the otic capsule, *4.* the otic capsule and the second visceral arch, and *5.* a "periarterial" aggregate of cells distinct from visceral or cranial

elements. The concept of a composite origin, partly from the second visceral arch and partly from the otic capsule, was first supported by Gradenigo,[13] van Noorden,[14] later by Hertwig,[15] Jenkinson,[16] Macklin,[17,18] Reagan,[19] and McClain.[20]

Bast and Anson, with their exhaustive studies, confirmed the origin of the stapes from these two sources: the visceral bar provides the annulus stapedialis; subsequent fusion with the lamina stapedialis of the otic capsule and secondary differentiation of laminar tissue provide the stapedial rim, the vestibular surface of the base and the basal perichondrium. The annular ligament develops through further modification of the peripheral laminar tissue.

With this concept of the embryology of the stapes, the observations described in this report find their explanation in an anomalous development. We are confronted here with an incomplete fusion of the annulus with the lamina stapedialis, and with a lack of further differentiation of the lamina stapedialis. As a result, the stapedial base is deformed, and no true annular ligament has been developed. Ossification of the cartilaginous otic capsule, therefore, brought about the osseous obliteration of the vestibular fenestra. Since no "window" has ever existed in this pair of temporal bones, in the sense in which the term fenestra is used to delineate the opening into the vestibule of a fully developed otic capsule, the term ankylosis is not appropriate to characterize this malformation. The conclusions are then that the stapes fixation is the result of a congenital lack of differentiation and development of the stapedial base and of its annular ligament. The presence of other malformations in the inner ear corroborate this interpretation.

SUMMARY.

Histopathological observations in a case with bilateral congenital fixation of the stapedial footplate are reported. The findings are in agreement with the clinical impression of the nature of the lesion. Failure of normal development of the footplate of the stapes, with an extensive persistence of the continuity of the lamina stapedialis (of Bast and Anson) with

the otic capsule, explains the condition found. The histologic sections show no evidence of an otosclerotic process.

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Section of Laryngology.

Program for session 1961.

February 3—Discussion: Recent Advances in the Treatment of Intrinsic Carcinoma of the Larynx.

March 3—Discussion: Chronic Sinusitis in Children.

May 5—Discussion: Vasomotor Rhinitis.

Section of Otology.

Program for session 1961.

February 3—Papers: 1. Problems of Tympanoplasty. 2. Experience with Pedicled Temporal Muscle Flaps in Radical Mastoid and Tympanoplasty Operations. 3. Is Myringoplasty Worth While?

March 3—Papers: 1. The Pathological Bases of Some Labyrinthine Vascular Disorders. 2. Meniere's Syndrome.

May 5—Paper: The Present Position of Stapes Footplate Surgery.

Members of the American Laryngological Association, the American Triological Society and the American Otological Society who will be in the United Kingdom at the time of any of these meetings will be very welcome.

STAPES SURGERY.

JUAN M. TATO, M.D.,

Buenos Aires.

In certain cases of otosclerosis Shea[1] removes the incus including the footplate; covers the oval window with a vein graft and inserts a polyethylene tube between the vein graft and incus. I developed a variation of Shea's procedure in which a mucous flap made from the mucosa of the promontory

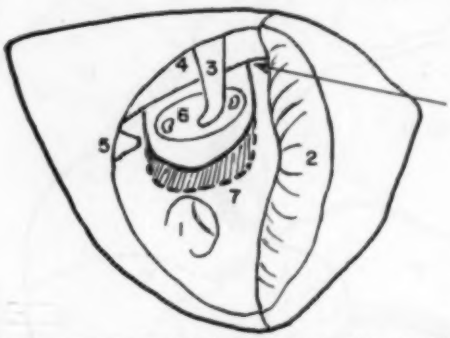

Fig. 1. Diagram of the surgical field seen after completion of the operation without the polyethylene strout. The arrow points to the starting point of the incision whose margin is indicated by a dotted line. The striped region corresponds to the gap between the incision and the flap which has been replaced. 1, round window; 2, tympanic membrane; 3, incus; 4, wall of facial canal; 5, eminentia pyramidalis; 6, oval window; 7, promontory.

and oval window niche is substituted for the vein graft. This procedure is as follows.

The head and crura of the stapes are removed. The mucosa of the promontory is cut with either a sharp point or round knife. The incision starts at the point where the cochleariform process makes contact with the wall of the facial canal and proceeds vertically downward until the level of

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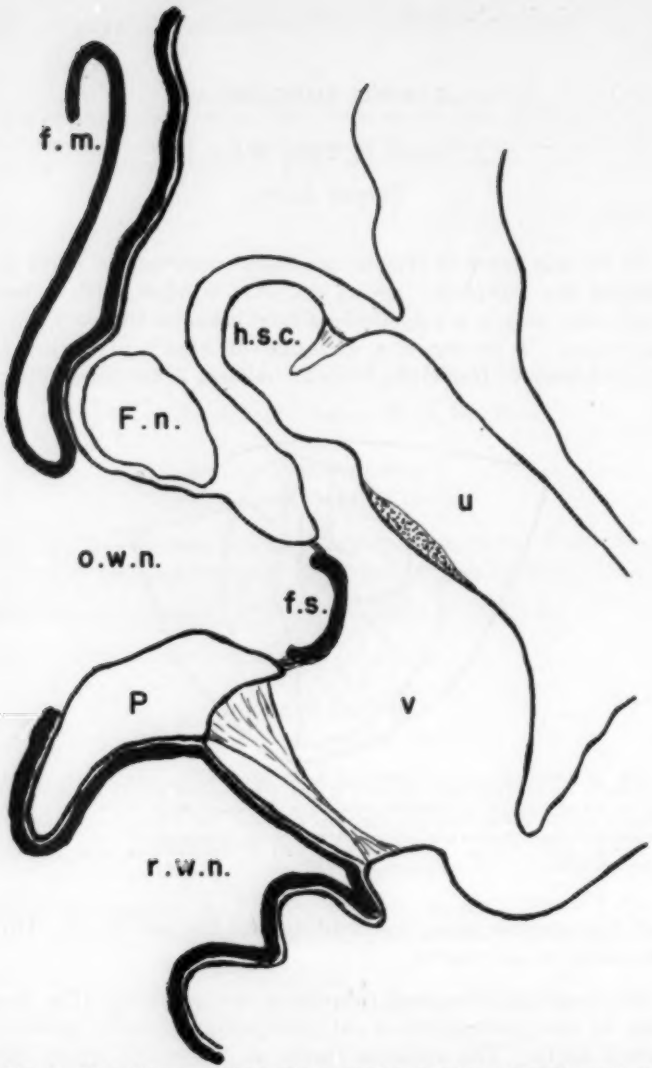

Fig. 2. This diagram represents a section passing through oval and round windows after the mucosa has been detached and elevated over the wall of the facial canal. The outline of this diagram was taken from a section of human temporal bone (40X). The head and crura have been removed but the footplate of the stapes still remains in place. In this figure and in Fig. 3, structures are identified by the following symbols: F.n., facial nerve; f.m., flap of mucosa; u., utriculus; o.w.n., oval window niche; r.w.n., round window niche; f.s., footplate of stapes; P, promontory; v., vestibule; h.s.c., horizontal semicircular canal.

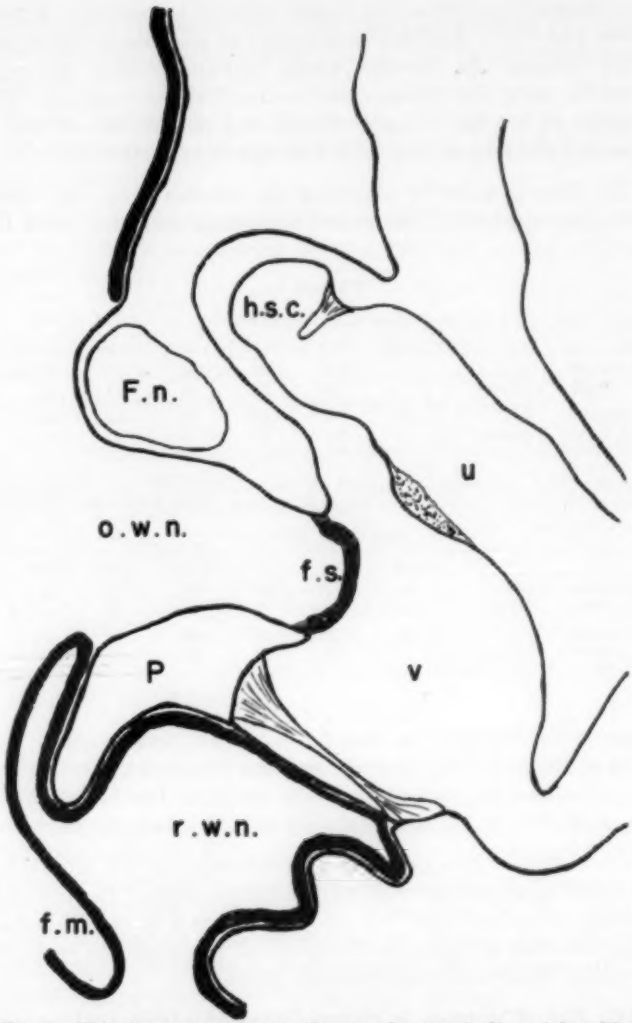

Fig. 3. This diagram represents the procedure used alternately with the U shaped flap. The transversal section is made along the wall of the facial canal and the flap turned over the promontory.

the superior margin of the round window is reached; it then turns posteriorly and proceeds as far as possible in this direction; finally, the incision turns upward, behind the oval window, until the facial canal is reached once again. The borders of the incision are shown as a dotted line in Fig. 1. Hemorrhage may be controlled by electrocoagulation.

The flap is made by elevating the mucosa from the transverse cut upward. This procedure successfully uncovers the

TABLE I.

Miss E. C., 44 years.

		128	256	512	1,024	2,048	4,096	8,192
Preoperation	Air		90	80	80	85	85	75
Sept. 2, 1958	Bone		20	20	25	30	30	
Postoperation	Air	15	5	10	15	20	60	50
July 30, 1959	Bone		15	10	5	15	35	

TABLE II.

Miss C. H. de G. D.

		128	256	512	1,024	2,048	4,096	8,192
Preoperation	Air	50	50	60	55	60	80	80
Aug. 1, 1958	Bone		0	0	0	5		
Postoperation	Air	25	20	20	20	30	35	60
Sept. 8, 1959	Bone		10	10	10	20		

promontory between the round window and oval window, the walls of the oval window niche and the footplate of the stapes. This flap is then turned over the walls of the facial canal. Fig. 2 illustrates the mucosal flap turned upward.

The U shaped flap can be made inversely, *i.e.*, the horizontal cut running along the wall of the facial canal. In this case the flap is turned over the promontory as Fig. 3 illustrates. After the flap is made, the footplate of the stapes is partially or totally removed in the classical manner.

The flap of mucosa is replaced over the bone surface and oval window niche. After replacement, a small gap remains between the margins of incision and the flap, as the striped region of Fig. 1 indicates. The gap is due to retraction of

mucosa after it is detached. The flap also presents two small openings which correspond to those of the crura which were removed.

Finally, as described in the literature, a polyethylene tube is inserted between the incus and the mucosa which lies over the oval window.

I have operated upon eight cases with this procedure. The best as well as the poorest results are here presented (see Tables I and II).

I think that the advantages of the mucous flap are clear when the results are compared with other procedures, *i.e.*, vein graft, covering the fenestra ovalis with gelfoam or leaving the oval window open with a prosthesis in place.

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ANNOUNCEMENT.

The University of Minnesota announces a one-day continuation course in Otolaryngology for Specialists which will be presented at the Center for Continuation Study January 26, 1961, preceding the Sectional Meeting of the American Laryngological, Rhinological, and Otological Society in Minneapolis on January 27-28, 1961. Guest registration for the meeting of the "Triological" will be welcomed. The program for the continuation course has been designed to complement the scientific program of the "Triological" Society.

Lodging and meals are available at the Center for Continuation Study.

VENTRICULAR DYSPHONIA: NEW INTERPRETATION OF AN OLD OBSERVATION.*

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PURPOSE OF THIS STUDY.

The science of optics is one of the oldest branches of physics. This may be one reason why eye specialists always devoted much time and study to the precise analysis of visual problems. Acoustics, however, made its greatest advances since the introduction of electronic techniques a few decades ago. Practicing otolaryngologists have little time to keep pace with the revolutionary development of psycho-acoustics and usually limit the evaluation of their patients' hearing losses to fundamental audiometry. Compared with the pure tone audiogram, the average laryngologist's description of a vocal anomaly is even more elementary, being restricted to a subjective judgment of the respiratory efficiency of the larynx and the social adequacy of the voice.

Ever since the invention of the laryngeal mirror, a few interested laryngologists devoted their efforts to the study of vocal pathology and collaborated with physiologists and acousticians in the clarification of vocal physiology. Known as *phoniatrists*, these specialists tried to promote the general understanding of vocal problems and assisted their surgical colleagues in the best care of their patients. Some also combined the practice of general otolaryngology and vocal rehabilitation. Unfortunately, the number of such phoniatrists

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at no time reached more than a few dozen in the medical centers of the entire world.

In order to meet this growing need in the area of communication disorders, a new profession has come into existence which is known as *audiology and speech rehabilitation.* Recruited from the ranks of educators, psychologists, and academic speech experts, the pioneers in audiology and communication sciences continue their outstanding contributions to these rapidly growing fields; yet, it is curious to note that vocal pathology attracts considerably less attention than do the questions of hearing and speech. As a rule, all abnormal voice conditions are treated by speech therapists under the vague heading of "voice problems."

As a result of these developments, the patient suffering from hoarseness may be handled from three entirely different viewpoints. The laryngologist is primarily concerned with the detection of malignant disease; the speech therapist is trained to teach desirable speech habits; the occasionally encountered phoniatrist combines laryngologic training and experience with the objective analysis of laryngeal function; moreover, these three specialists use different professional languages for the expression of their opinions which causes constant misunderstandings among these important representatives of sciences dealing with human communication.

The purpose of this paper is threefold: to show how well the old authors understood various diseases and disorders; to demonstrate the need for complete medical, laryngologic, as well as psychologic evaluation in all cases of vocal pathology if the patient is to be considered a whole personality (and not an assembly of specialized interests); and to offer a theory for the psycho-dynamic explanation of hyperkinetic voice disorders as a phenomenon of regression in response to stressful situations.

HISTORICAL REMARKS.

It is now exactly 100 years since J. N. Czermak[11] observed for the first time that phonation with the ventricular folds is a pathologic phenomenon. Five years after the invention of

the laryngeal mirror by Garcia (1854), the two fathers of laryngology, Tuerck and Czermak, were well engaged in presenting their new findings to the medical profession; thus, the discovery of ventricular dysphonia was one of the first clinical descriptions resulting from the use of laryngoscopy.

Further developments in the knowledge of the function of the ventricular folds for phonation were studied by Panconcelli-Calzia.[30,31] He showed the illustrations of various authors which clearly depict the hyperkinesis of the ventricular folds, for instance those of Merkel[25] (1862). In his textbook on laryngology in 1893, Moritz Schmidt[28] described the faulty phonation with the ventricular folds. A systematic study of the ventricular folds was presented by Flatau and Gutzmann[16] in their book on ventriloquism (1894). Flatau[14,15] also dealt with therapy of ventricular dysphonia (1910, 1928). Further contributions to this subject were made by many authors as listed in the bibliography.[5,9,12,13,17,22,26,27,28,39,40,41] During World War I various laryngologists described their observations of ventricular dysphonia among soldiers. Similar experiences during World War II were reported by one of us.[3]

DEFINITION.

Ventricular dysphonia is known under many names: Taschenfaltenstimme (German), Voix des bandes ventriculaires (French), Dysphonia plic*ae* ventricularis,[23] Dysphonia plic*a* ventricularis,[37] Dysphonia plicarum ventricularum,[31] Dysphonia ventricularis,[43] etc. It is caused by the faulty participation of the ventricular folds (false cords) in the act of phonation. Normally, the ventricular folds represent the middle laryngeal sphincter and contribute to the firm closure of the larynx during deglutition and expectoration; hence, they belong to the *protective sphincter mechanism* of the larynx.

It is fascinating to compare Furstenberg's[18] experimental findings on the neural mechanism of laryngeal closure with the clinical signs of ventricular dysphonia. From such viewpoints we may now explain this voice disorder as the intrusion of a subcortical *primitive mechanism of phonation* after the regular cortical phonatory system had become inhibited by the

unconscious aggressiveness of an imbalanced personality as a reaction to the threats of daily life. This primitive neurolaryngeal mechanism can even be seen with the laryngeal mirror which shows the pathognomonic *sphincter action* of the laryngeal structures during all types of hyperkinetic phonation (see Fig. 1). Since all primitive laryngeal reflexes, like coughing, gagging, retching, vomiting, and swallowing, are characterized by the sphincter action of the epiglottis,

Fig. 1. Laryngoscopic image of hyperkinetic dysphonia with marked laryngeal sphincter action. Note the descent of the epiglottis, the anterior over-adduction of the ventricular folds, and the posterior deficiency of vocal cord closure.

Fig. 2. Laryngoscopic image of ventricular dysphonia. The ventricular folds are over-adducted during all phonatory actions. Often, this is combined with the laryngeal sphincter mechanism shown in Fig. 1.

cords and ventricular folds, this invasion by the primitive sphincter action of the highly specialized functions of human phonation indicates a regressive phenomenon.[4,7] It is interesting to note how psychiatric theory interprets this symptom of laryngophonic regression in the light of the projective test analyses of such patients.[20,21]

CLASSIFICATION.

Under the influence of the primitive laryngeal sphincter action, phonation with the ventricular folds (see Fig. 2) may originate from several somatic and psychogenic causes.[29] According to our description,[3,7] one may differentiate six specific forms of ventricular dysphonia, namely the habitual, emo-

tional, paralytic, cerebral, cerebellar, and the vicarious type, which are readily distinguished by history and examination.

1. Most frequently, it represents the extreme end stage of hyperkinetic dysphonia due to constant vocal abuse which is usually of *habitual* origin.

2. In times of general stress, or during a crucial period in the life of a psychoneurotic person, an emotional crisis may precipitate the psychogenic or *emotional* form of hyperkinetic dysphonia. This is always characterized by hyperkinesis of the ventricular folds.

3. The *paralytic* form is due to a rare compensatory reaction to laryngeal paralysis.[6] The ventricular folds take over the function of phonic glottal closure, substituting for the true cords which cannot meet due to the paralysis.

4. As a sign of dysarthria resulting from *cerebral* disease, the voice may change to an extremely low, choked, rough, intermittent, spastic, and peculiarly squeezed sound.

5. Spastic and labored phonation is most typical for *cerebellar* lesions, as described by several authors.[3,24] Mid-brain disease, such as in Parkinsonism, may also produce ventricular dysphonia.[24,31]

6. Finally, we know the *vicarious function* of the ventricular folds when they are substituting for the defective vocal cords. The systematic development of a well compensated ventricular fold voice is one of the goals of vocal rehabilitation after the loss of one or both vocal cords as a result of traumatic injury or surgical operation.

J. S. Greene[19] discussed the differentiation of the usual types of psychogenic dysphonia and aphonia, which are often referred to as being of hysterical origin, and the specific condition of spastic dysphonia, or psychophonasthenia, as he preferred to call it. It had been observed for a long time that emotional (or hysterical) aphonia and dysphonia exist in two main types: the hypokinetic and the hyperkinetic.

The *hypokinetic* type of psychogenic dysphonia is caused by incomplete vocal cord adduction during phonation. As a

result of the deficient or totally absent glottal closure the voice is reduced to a breathy and feeble sound (dysphonia) or to a faint whisper (aphonia). This is the form of emotional aphonia usually encountered in psychoneurotic or hysterical women.

On the other hand, the *hyperkinetic* type of psychogenic dysphonia shows the sphincter action of the laryngeal introitus. During his visibly great speaking efforts the patient tries to squeeze out his voice by forcefully overadducting the ventricular folds. This emotional type of ventricular dysphonia is distinguished from the specific disease entity of spastic dysphonia by the fact that the former affects also the singing voice, is always present, does not fluctuate between clear parts of a sentence and other words spoken with choked spasticity, does not divide the stressed vowels, and that the patient does not exhibit much anguish about his condition. These differences were analyzed in a previous monograph[3] on traumatic voice disorders and in a recent discussion of spastic dysphonia.[7]

DIAGNOSIS.

With a complete history and after careful laryngoscopic inspection, the patient's complaints can be readily evaluated. Examination of the *upper airways* reveals normal conditions. If some abnormal findings are encountered, they represent coincidental complications which have nothing to do with the habitual or psychogenic vocal disorder; however, the constant vocal strain may lead to secondary signs of tissue irritation. One of us[1,2] described how the typical findings of chronic laryngitis may very well reflect the *results* of hyperkinetic types of hoarseness, and not their *causes*. For this reason we often see a moderate redness or congestion of the vocal cords in cases of ventricular dysphonia. The constant overuse of the sphincteric action of the ventricular folds can even lead to their hypertrophy.[7]

Bruno[10] demonstrated the *hypertrophic appearance* of the ventricular folds on tomographic radiographs. It could also clearly be seen how the ventricles of Morgagni were obliter-

ated. This radiologic appearance did not change with various therapeutic efforts, including local anesthesia of the pharyngeal structures. In view of the recently renewed interest in contact ulcers of the vocal processes it is interesting to remember that Oltuszewski,[28a] in 1885, described two cases of contact ulcers which had developed as a result of prolonged spastic phonation. All these signs of *secondary laryngitis*[2] must not be misinterpreted as a local organic cause of the presenting vocal disability.

The patient's *hoarseness* is fully explained by the ventricular hyperkinesis and the visible sphincter action of his larynx. Such voices sound extremely low, harsh, and coarse, their basic pitch often reaching the "subtonal" frequencies between 50 and 100 cps. If the patient complains of *pain* in the throat, especially after having spoken for a while, his vocal overexertion and the faulty use of his larynx readily explain this complaint as a sign of functional irritation.

Often, we hear of a *lump* in the throat, especially below the larynx. According to Tremble,[42] this complaint is frequently due to esophageal spasm. Similar spasm of the m. cricopharyngeus is also due to nervous tension. Such signs will direct one's attention to some underlying emotional factors. In case of remaining doubt a frontal tomograph will bring final clarification. Once the precise type of ventricular dysphonia has been recognized, therapy is directed by the individual findings.

CASE REPORTS.

Case 1. White female, age 33, married housewife, complained on October 10, 1957, that she suddenly lost her voice three weeks previously.

Previous History: Recurrent bronchitis, once diagnosed as asthma. First marriage at age 19, divorced after one year, second marriage one year later. Seven pregnancies, one uterine curettage, two miscarriages, five children are alive and well. Had numerous surgical operations: appendectomy (age 17), ovarian cyst (22), tonsillectomy (23), D and C (24), hysterectomy (30), excision of benign tumor from left axilla (31). She was always very nervous and unstable.

History of Vocal Complaints: When 16 years old, she was rehearsing for a school drama performance. During some dispute she fainted and awoke voiceless. After four weeks of whispering she regained her voice and could perform in the leading role as she had aspired. Ever since she experienced occasional attacks of sudden hoarseness. Often, she felt a choking sensation in her throat, as if a hair or a lump were stuck. For

the past few months she had lived with her mother while her father had a kidney operation and suffered his fourth stroke. Some of her five children were also ill. Three weeks ago, one of her children came up to her bed at 3 a.m. and wanted to sleep with her. She felt angry, tried to send the child to his own bed, but could not utter a sound. Next morning she was still voiceless and consulted her family physician who gave her a sedative. Various other ataractics were subsequently prescribed without effect on the voice.

Examination showed a well-developed young-looking woman. Ears and the upper airways showed normal findings. Larynx: the vocal cords were slightly congested and moved normally; however, during phonation they were completely covered by the over-adducted ventricular folds. The voice was totally aphonic and sounded like a harsh whisper.

Fig. 3. Anterior laryngeal tomograph of ventricular dysphonia. Note laryngeal sphincter action with closure of ventricular folds and lowered epiglottis (Case 1).

Radiologic Analysis: Lateral radiographs of the neck as well as frontal tomographs during quiet respiration were normal; however, tomography *during phonation* showed characteristic changes. Fig. 3 demonstrates a frontal section through the anterior half of the larynx. Cords and ventricular folds are firmly approximated, Morgagni's ventricles were compressed, the epiglottis was lowered, and the trachea was moderately widened as a sign of high subglottic air pressure. Fig. 4 is a frontal section through the posterior half of the larynx. Again, cords and ventricular folds are completely closed. Morgagni's ventricles appear as narrow slits. The arytenoids are in normal adducted position. The trachea is moderately widened by high subglottic pressure. For better comparison, a normal tomogram is shown in Fig 10.

Diagnosis: Psychogenic ventricular (hyperkinetic) aphonia, symptomatic of conversion hysteria.

Course: Under verbal persuasion, the patient was able to produce several clear vowels; however, she immediately reverted to her harsh and forced whisper. Obviously in need of psychiatric treatment, she was referred to our neuro-psychiatric clinic for evaluation and psychotherapy. A few months later she regained her voice overnight.

Case 2. White male, age 58, salesman, presented himself on February 23, 1957, with the complaint of hoarseness and pain in his throat. His voice had weakened for several years, especially at times when he talked much. No major illnesses noted.

Examination showed moderate enlargement of the lower nasal turbinates. Tonsils were absent. The house surgeon observed moderate

Fig. 4. Posterior laryngeal tomograph of ventricular dysphonia. Note compression of ventricles, closure of ventricular folds, and dilatation of trachea (Case 1).

hyperemia and congestion of both vocal cords which did not close perfectly over the posterior portion of the glottis. The voice sounded hoarse and rough. The patient gave a calm and well adjusted impression.

Impression: Chronic laryngitis was assumed, and a course of antibiotics and expectorants was prescribed.

Course: A week later, the patient returned with the same complaint and unchanged findings. He was advised to use steam inhalations and rest his voice. After another week, he was referred for vocal analysis. This time it was noted that his larynx presented typical changes. The ventricular folds appeared hypertrophic and abnormally mobile. The vocal cords were plump and congested. Observation of phonation revealed the following peculiarities: at the onset of phonation the vocal cords closed normally, but the ventricular folds began to bulge over their mid-

portions. This stage was followed by complete closure of the ventricular folds which visibly vibrated, while a low and rough voice was audible. At the end of phonation, the ventricular folds began to separate anteriorly and posteriorly until shortly afterwards their central bulge disappeared. This cycle was concluded by abduction of the vocal cords. Fig. 5 demonstrates this sequence of ventricular fold hyperkinesia. The voice sounded unusually low, hoarse, rough, and harsh. Average speaking pitch was found around tone E (82.5 cps), the vocal range appeared restricted to a few tones, and phonation time was shortened to 8 sec. (vowel "o" on tone E).

Diagnosis: Habitual ventricular (hyperkinetic) dysphonia resulting from occupational abuse of the voice.

Therapy: After ascertaining that all other clinical and laboratory findings were normal (sinus X-ray, CBC, serologic, blood sugar, urine, etc.),

Fig. 5. Sequence of laryngeal movements during one cycle of phonation in a case of ventricular dysphonia. I: respiration; II: onset of phonation with beginning hyperkinesis of ventricular folds; III: phonation with ventricular folds, note narrowing of laryngeal introitus; IV: beginning relaxation of ventricular folds towards end of phonation; V: beginning separation of vocal cords at end of phonation, note elevation of epiglottis (Case 2).

the patient was counselled about the proper use of the voice. He was advised to speak little and in a relaxed manner and was shown how to do some vocal exercises. Steam inhalation and an antihistaminic were suggested for symptomatic relief.

RESULTS.

On April 2, 1957, the patient reported great improvement and spoke with a clearer and higher voice. His larynx appeared less irritated and the hyperkinesis of the ventricular folds was reduced so that regular vibration of the vocal cords could then be seen. In the course of the next months the patient continued to improve and was able to speak with a fairly clear, loud, well placed voice. Laryngeal congestion had disappeared, and the hyperkinetic movement of the ventricular folds was greatly diminished.

Radiographic analysis was undertaken at the beginning and end of this course of vocal rehabilitation. Whereas the lateral

view of the larynx was always normal during quiet respiration (see Fig. 6), the tomographs taken *during phonation* demonstrated the ventricular hyperkinesia. In the stage of marked ventricular dysphonia (see Fig. 7), the vocal cords and ventricular folds were seen in close approximation, obscuring the ventricles of Morgagni. The trachea appeared markedly

Fig. 6. Lateral laryngeal radiograph during quiet respiration in a case of ventricular dysphonia, showing normal structures and positions (Case 2).

dilated as a result of high subglottic pressure. Since the return of more normal phonatory movements (see Fig. 8), the cords close well, and the ventricular folds remain sufficiently apart to allow normal phonation.

Case 3. White male, age 60, salesman, attended the N. Y. Eye and Ear Infirmary for several years. In May, 1958, he complained of increasing

hoarseness which began six months before. He does not smoke, but talks much, and loses his voice when he gets excited. He sells ladies' hats and is disturbed by lint and dust.

Previous History: Appendectomy age 13, typhoid fever age 25, tonsillectomy age 45. Had chronic sinus trouble requiring several nasal polypectomies and sinus irrigations. For the last five years he suffered from gastric ulcer, treated with diet and medication.

Examination revealed a depressed man, looking older than his age. The nasal mucosa had the so-called allergic appearance, and several small

Fig. 7. Frontal laryngeal tomograph of ventricular dysphonia. Note compression of ventricles, over-adduction of ventricular folds, and dilatation of trachea during faulty phonation (Case 2).

polyps were seen in both middle meatus. Sinus X-ray showed cloudiness of antra and ethmoids. The vocal cords were red, dry, swollen, and hypertrophic. The ventricular folds appeared hypertonic and closed completely during phonation, thus obstructing the true cords. Accordingly, the voice sounded very rough and low, with an average speaking pitch around F (87 cps) and a short phonation time (o/F = 6 sec.).

Psychological Evaluation: Has been married since 1920, two sons are married and well. His wife is nervous and excitable. One year ago his daughter-in-law developed a psychosis following childbirth. She has fits of depression, indulges in compulsive rituals, cries often without reason

and refuses to take care of her baby which had to be placed in the patient's home. Her visits in his apartment usually produce quarrels, so that he refuses to see her. He also had several financial losses, during the depression, after the last war, and six months ago when some speculative stocks failed. He had bought them five years ago, raising the money by selling his house. When confronted with the coincidence of his gastric trouble with the investment in these speculative stocks at the price of his house, and of the onset of dysphonia when he lost his money, the patient admits the emotional element of his ailments. In conclusion,

Fig. 8. Frontal laryngeal tomograph of ventricular dysphonia. After improvement of the faulty phonation pattern, the vocal cords close well, the ventricular folds are less approximated, and the laryngeal introitus remains wider (Case 2).

this man states that he is far from satisfied with his life, deplores the loss of his economic security by imprudent speculations, and resents the necessity of visiting a hospital clinic.

Diagnosis: Psychogenic ventricular dysphonia associated with other psychosomatic disorders (allergy, gastric ulcer), resulting from a tension-anxiety state.

Radiologic Analysis: Frontal tomographs taken during *phonation* (see Fig. 9) show marked hypertrophy of the vocal cords and of the ventricular folds. Both are firmly closed. The ventricles of Morgagni appear as faint slits. That on the right side is seen on a lower level than on the left which is not explained by the slight tilting of the thyroid cartilage

to the right side. The trachea is markedly widened by high subglottic pressure.

Treatment had to be confined to antiallergic medication, symptomatic care of the throat, and brief counselling on the management of his voice. Since the patient was too preoccupied with his financial and domestic problems, he refused a course of vocal rehabilitation and was also disinclined to seek psychiatric care.

Fig. 9. Frontal laryngeal tomograph of ventricular dysphonia, showing compression of the ventricles, firm closure of the hypertrophic ventricular folds, and dilatation of the trachea (Case 3).

TREATMENT.

In all cases of vocal disability due to disorders of function, the choice of appropriate therapy depends on the individual somatic and emotional findings. Obviously, the six types of ventricular dysphonia require different management.

Vocal rehabilitation, based on the judicious application of orthophonic principles, is indicated in the majority of cases which present the habitual type resulting from constant vocal misuse. Providing the patient demonstrates sufficient moti-

vation for his share of the rehabilitational effort, satisfactory results can usually be achieved. The necessary techniques of vocal re-education must be studied in theory and practice for they cannot be improvised.

Psychotherapy, either by the expert psychiatrist and clinical psychologist, or offered in a supportive manner by the experienced phoniatrist, represents an integral part of the rehabilitation program. It is of primary importance for the *emotional* type of ventricular dysphonia.

Physiotherapy may be helpful in selected cases, especially those of the paralytic or central types. This includes vibratory massage of the neck for the reduction of muscular overcontraction; heat for improved circulation, or for reduction of subjective complaints such as pain, tension, or globus sensation; and the special techniques of isochronic tone massage which aid laryngeal compensation by promoting the auditory, tactile, and kinesthetic pathways for homeostatic feedback; however, electric stimulation is usually contraindicated because it tends to augment the neuro-muscular hyperkinesis.

Local laryngeal procedures may be used in a variety of techniques. Flatau[15] and H. Gutzmann, Jr.,[19a] advocated the use of a special spreading forceps. After local anesthesia, the branches of the forceps are introduced between the overcontracted ventricular folds, spreading them apart to allow free vibration of the vocal cords. Some patients then learn to maintain the re-established function of the cords. Rethi[32-36] prefers novocain injection into the palato-styloideal fold of the stylopharyngeus muscle by the endoral route. The value of this method was recently confirmed by Beckmann.[8] Sometimes, simple surface anesthesia of the larynx may be sufficient to break the faulty pattern of phonation.

Surgical procedures should be reserved for obstinate cases. Jackson and Jackson[23] recommended the excision of the hypertrophic portion of the ventricular folds. Rethi[35,36] proposed the surgical excision of the stylopharyngeus muscle by the cervical approach.

While all these techniques serve for the abolition of the

Fig. 10. Frontal laryngeal tomograph of a normal male (baritone), while humming a high tone in head register. Note the sharp edge of the cords, the wide open ventricles, the abducted ventricular folds, and the wide supraglottic resonance chamber.

undesirable types of ventricular dysphonia (types 1 to 5), the *vicarious type* is systematically promoted to replace vocal cord function after it had been lost by trauma or surgical intervention. The development of vicarious ventricular voice is based on the techniques of orthophonic re-education and may be assisted by physiotherapy as mentioned above. Further details were discussed in previous works.[3,24] Surgery can be of service when protruding scars or granulations need ablation for smoothing the contours of the phonating parts.

Prognosis is usually excellent when the therapeutic situation is satisfactory. Therapeutic results were analyzed in a pre-

vious monograph.[3] Occasional failures are due either to diagnostic errors, or to the resistance of deeply neurotic patients. Therapeutic progress may be corroborated by radiologic control which should show a return to more normal conditions (comp. Fig. 10).

CONCLUSION.

Although the phenomenon of phonation with the ventricular folds has been known for a century, and although careful studies of ventricular dysphonia were submitted by various authors during the last 50 years, this frequent disorder of the speaking voice is usually neglected in clinical practice. The faulty habit of using a primitive laryngeal mechanism for self-expression and communication produces secondary organic changes of laryngeal structure. Often, these visible alterations of laryngeal appearance (secondary or traumatic laryngitis) are misinterpreted as the *cause* of the vocal disability, while in reality they represent a secondary *result* of the primary vocal disorder.

In the light of present knowledge, all types of hyperkinetic phonation reflect a regressive psycho-dynamic phenomenon. The underlying primitive sphincter mechanism of the larynx may be activated by secondary subcortical centers of phonation. These can take over the function of the primary cortical centers of phonation when the latter become inhibited by the unconscious withdrawal of an emotionally maladjusted personality to the more primitive mechanisms of neurotic aggression and hostility.

Since, then, the "laryngitic" appearance of a larynx traumatized by ventricular dysphonia must be differentiated from true infectious laryngitis, such patients cannot be treated successfully, unless the actual underlying cause of their vocal disability has been ascertained. This differential diagnosis between true and secondary laryngitis should consider the six main types of ventricular dysphonia, namely the habitual, emotional, paralytic, cerebral, cerebellar, and the vicarious forms.

SUMMARY.

The frequent problem of ventricular dysphonia is briefly discussed by first surveying the large amount of pertinent literature. The laryngoscopic findings of secondary laryngitis must be distinguished from true laryngitis. Correct diagnosis is established by history, analysis of laryngeal and vocal function, and consideration of the patient's personality.

Since ventricular dysphonia occurs in at least six types, treatment depends on the underlying cause. Three typical case histories illustrate the usual findings. Laryngeal tomography is valuable for the demonstration of hyperkinetic laryngeal function. The theory is advanced that laryngeal sphincter action during phonation represents a phenomenon of laryngophonic regression which the psychiatrist can utilize for his psycho-dynamic interpretation of the patient's disturbed behavior.

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THE PSYCHOLOGICAL MANAGEMENT OF THE SURGICAL PATIENT: VOODOO, MESMER AND ALEXANDER POPE.*†

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INTRODUCTION.

The subtitle of this essay contains three items designating the headings: Under Part 1, Voodoo, I will attempt to convince you of the basic reality that psychological factors can have profound influence, including physical effects, on the surgical patient—really all patients—and the practice of surgery. For example, Walter Cannon, the famous physiologist, collected a number of well-authenticated cases in the literature under the title "Voodoo Death." Individuals *believing* themselves hexed or hoodooed have died in a matter of hours or days without discernible organic explanation. Of course, death by whatever cause ultimately is organic or physical; however, the reasons for death and the mechanism in the cases above are unknown.

Part 2, Mesmer, will be concerned with the personality of the physician, and particularly the physician's beliefs or convictions, as one of the most important influences, for better or worse, on the patient.

Part 3 is an elaboration on two lines from the work of the poet Alexander Pope:

> *"Know then thyself, presume not God to scan;*
> *The proper study of mankind is man."*

The admonition "Know thyself" has been ascribed to at least six other people, but Pope's stanza best suits my purpose;

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also pertinent is the proverb "Physician, heal thyself," a reference which St. Luke ascribed to Jesus. Granted the fact that profound influences are possible by psychological methods, and that the physician is, himself, strong medicine, then both quotations summarize my thesis—that if a physician is to use his medical knowledge most effectively in his practice, he must really understand himself. Knowing himself is the most difficult part, but it is the cornerstone of any intelligent, rational, and effective use—and explanation—of psychological principles in the management of *any* patient.

PART 1—VOODOO.

Many physicians pay lip service to the concept, even truism, that psychological factors are important in the general practice of medicine, or at least will acknowledge particular situations in which it is important; yet, their conviction is usually not very strong or is considered inapplicable to their own practice. For example, if a patient has a headache which benefits from a "sterile hypo" or some other placebo, the implication is that the headache was not real, that is, was due to "psychological factors only." A placebo can affect the headache caused by cancer as well as the one caused by nervousness or tension. Just as a placebo can relieve pain of organic origin, so can it alone produce marked physical changes and alter physical illnesses. Good examples of this are found in Stewart Wolf's article[1] on marked toxic reactions from placebos, including urticaria, generalized collapse and a disseminated dermatitis. Whatever an individual's illness, the illness and the patient can be conceived of only in terms of complex psychological, physiological, and environmental factors. As an example of this last statement, think of a not necessarily hypothetical case in which a man and his wife have a heated argument resulting in his leaving the house and visiting several bars. He becomes intoxicated, wanders about the streets in freezing weather—and ultimately develops pneumococcus pneumonia and a lung abscess requiring surgery. In such a case, what would you say caused the pneumonia and the abscess? Psychological factors are most often just one facet of the genesis of the illness or its manifestations.

Few physicians doubt that such things as Christian Science, or some other strong belief similar to Voodoo, can affect people with functional or psychological problems; however, most doubt that those who have a "real illness," an organic problem of some sort, can be helped by so-called mystical, superstitious, or unscientific influences. If the effect of such influence is acknowledged at all, it is considered pertinent only in the sense that psychological problems can make an organic illness worse. I am convinced that whatever one believes in strongly enough can produce functional and physical changes both in the direction of pathology—disease—as well as in the direction of recovery—health. Although my personal experience with Voodoo is limited at this time, our study of Voodoo here in Mississippi would confirm my impression.

I have used various forms of suggestion and/or hypnosis for years in the treatment of warts with excellent success. I started to use this method after I had read an article in 1948 by a dermatologist on the treatment of warts with such measures. I remember well that my grandfather used to bury pennies, and folklore is full of a variety of successful methods, to remove warts. Although warts are considered of virus origin, in two recent articles Ullman[2,3] documented the fact that in many clearly discernible instances the resolution of warts can be brought about by psychological means such as suggestion or hypnosis. Along this same line, Dubois said repeatedly that the tubercle bacillus, although *necessary*, may be one of the least important factors in the patient with tuberculosis; and this includes etiology.

Many experienced surgeons and anesthetists will freely admit that the patient who does not wish surgery or who feels that something "bad" is going to happen is a poor risk, particularly if his convictions seem fixed and strong. Some of these patients die without adequate explanation. I am reminded of several such cases which I encountered during the Korean War. In one instance a soldier was brought in who had a shoulder wound of no great consequence but who had no desire to live. His injury occurred during the Winter of 1950, when the fighting was particularly fierce, and there was

little opportunity for psychiatric treatment for the man. In a matter of days he died, and autopsy revealed no adequate physiological cause of death. Additional proof of the power of the mind lies in the fact that thyroidectomies, hysterectomies and other operations can be performed with no anesthesia whatsoever other than hypnosis.

If I have seemed to belabor the obvious, forgive me, but ten years' experience teaching medical students and graduate physicians has convinced me that there seems to be little individual personal conviction or understanding, and rarely utilization, of such truths.

PART 2—MESMER.

In the preceding section the importance of psychological factors, *per se*, and particularly the patient's belief and conviction have been emphasized. The secret of whether voodoo will work lies in the fact that the individual who is hexed believes firmly in hoodoo and expects results therefrom. The "conjurer-doctor" need not believe in hoodoo, but he must believe that it will exert a change, that it will influence the individual. In other words, he doesn't need to believe it works by reason of magic, but he must expect results. In this section I will be concerned with the physician's beliefs and convictions and their influence on the patients, and with the importance of the setting or trappings—call them stage effects if you like. Mesmer and his career very nicely illustrate these concepts. One other point—as wrong as he was about the how of what he was doing and the theory behind it, he was very right about the fact that it worked well; thus, he was very successful in its application.

Franz Anton Mesmer lived from 1734 to 1815. His doctor's thesis in 1756, when he graduated, was on "The Influence of Planets." He believed that it was possible to effect cures with a magnet through the mechanism of what he termed animal magnetism. Mesmer carried out his treatments in a darkened room where a group of people were seated around a large tub or baguet, from which iron bars protruded. By his touch, he professed to charge these bars with animal mag-

netism, which flowed through tne circle of people when they grasped the bars. Mesmer would appear in a lilac robe carrying a wand. He would touch the baguet with the wand and immediately the people in the circle would become agitated and begin to twitch and writhe. Mesmer's treatment was called mesmerism and it soon became synonymous with hypnosis. One of Mesmer's disciples, Puységur, rediscovered hypnotism, which in one form or another has been known for many centuries. Mesmer has been dubbed a charlatan or quack, which I think is distinctly unfair, because he was a physician and went through the conventional training of that day and age. After all, our medical colleagues espouse and employ many so-called scientific theories which are basically incorrect, yet this fact, *per se*, does not make them charlatans. To be sure, Mesmer's explanation of his treatment-method was incorrect, but no one who wishes to study his influence on that day, and no less upon succeeding generations, can deny this influence.

While I am on the subject of quackery, I would like to state my belief that in some ways we in medicine are responsible for the success of present-day charlatans or quacks. We are too dependent upon science, and we belittle faith and psychological influences. Charlatans can and do *help* people suffering from all kinds of ailments and illnesses, both organic and functional—and I state this categorically. The danger is that these healers are untrained and unable to know what they cannot help or when they are not helping. Consequently, precious time may be lost, as for instance in such conditions as cancer. Another fact is very important here: namely, for many people it is much more acceptable to have, for example, the chiropractor's adjustment for whatever ails them rather than to face the fact that theirs may be a psychological or psychiatric problem, particularly if referral to a psychiatrist may follow; yet this not unexpected attitude toward psychiatric referral is precisely why all physicians must be able to apply many psychiatric principles and practices. The only resources the charlatan has are psychological. The physician can utilize the same psychological resources *plus* all available scientific knowledge and methods for treating all facets of most ill-

nesses, *plus* the prestige of the medical profession. When necessary the psychiatrist can be utilized for advice, consultation, or referral. Skillful referral is so important I could spend my whole time on the subject. Discuss this with those psychiatrists to whom you refer patients.

Sadly, doctors seldom miss the physical or organic facets of illness but badly neglect the others. Kaufman and Bernstein[4] documented this point in rather convincing fashion. Mount Sinai Hospital has a diagnostic clinic for local physicians' problem patients whose income is between $5,000 and $8,000. For a flat fee the patient receives all the studies necessary, including laboratory work and consultations with various specialists. All facets of medicine are represented, including psychiatry. In 1,000 unselected patients studied in this clinic 81.4 per cent had diagnosable psychiatric syndromes, and in over one-half of this 1,000 patients, the psychiatric trouble was the major or exclusive problem. As psychiatrists, Kaufman and Bernstein echo the refrain "Emotional illness can and should be a positive diagnosis, not one of exclusion." It should be made by positive criteria rather than being the only diagnosis left when everything else is ruled out.

To summarize this part then, let me emphasize that the physician is by far the most powerful medicine available today for the patient. The old statement that the "operation was a success, but the patient died" may be nearer the truth than we like to think. For in surgery there are many, many excellent technicians with superior operating skill. The really great surgeons are also great physicians, albeit often unconsciously, I regret to say. That is, many of them have tremendous personalities which greatly affect their work and their patients. Sometimes, like Mesmer, the results are good, but the physicians' analyses of why are incorrect. Many of them would be ashamed to use a placebo but will give this or that favorite prescription to the patient with the expectation that the patient will benefit from it—and the patient does; but in many cases the improvement had little to do with the medicine given.

Stewart Wolf[5] illustrated this point in the field of surgery. Wolf, Professor of Medicine at the University of Oklahoma,

discussed a surgical meeting in the 1930's where two surgeons were reporting on their experience with gastroenterostomies. Dr. D, who was very much in favor of the operation, reported favorable results with the recurrence of marginal ulcers in only 1.6 per cent in his series. Dr. B, who had abandoned the procedure and had never been much in favor of it, reported that from the observation in his series there was 33 per cent incidence of marginal ulcers. The only explanation possible for this much difference is the personal beliefs of the individuals and the influence of those surgeons on their patients. At this point it would be well to reiterate Dr. Wolf's admonition, which I endorse enthusiastically, that in emphasizing the power of the doctor-patient relationship we be fully aware that it isn't the *only* good treatment and that it is only one facet in most cases.

Harley Williams,[6] an Englishman, wrote a book called "The Healing Touch," a series of biographical sketches on various physicians. On the jacket of the book there is a statement that "The thread running through this book is that of personality." The author has this to say, "At heart, we all believe in the healing touch. It is no mere sentimental phrase but a powerful reality. We may not possess this gift, but we have certainly experienced it at the hand of others. How are we to define it? Something nearer magic than we are likely to admit. A sort of magnetic force in the personality that puts fear to rest, releases the restless currents of our being and harmonizes them in a new direction. Some people have it, the majority do not. This powerful human faculty is felt—in ordinary affairs, in politics, in business, but its most natural place is in the ancient art of healing, and in the medicine and surgery that have grown therefrom. It is fundamental today just as in the simpler medicine of a previous age."

Apropos of this last statement, the observation has been made that when doctors could cure only a few ailments they were often held in high regard; now in an age of therapeutic excellence they often have to defend themselves. The possible explanation for this is that physicians and surgeons have become so dependent upon the miracle drugs and their vast

technical accomplishments that they have neglected the interpersonal and cultural factors in the practice of medicine, that which is often called the art of medicine. I am not in complete agreement with this latter designation, the art of medicine, for I think that it is coined to explain in somewhat mystical fashion why some doctors are so adept and successful in their medical or surgical practice. I believe that there are more scientific explanations for their success with their patients.

PART 3—ALEXANDER POPE.

To repeat, "Know then thyself, presume not God to scan;/ The proper study of mankind is man." If you will accept the fact that your personality vastly influences the treatment and results you get, then some other things follow. It is incumbent upon all of you to know as much about yourself as you possibly can. This does not mean psychoanalysis, nor does it require any really time-consuming action; rather, it requires a willingness to know yourself, and the knowledge that to do so is most important. Consider such questions as: Why do some patients irritate you and how do you respond to certain patients? What can you say about the way and the manner that you practice medicine and your approach to patients? What kind of "bedside manner" do you have and are you reasonably sure that you are making the most effective possible use of yourself? What kind of conditions and diseases can you treat best, and with which do you have the best results? The answers to these questions and the process of such scrutiny could and should improve the efficacy of your practice considerably. To suggest that the de-sensitization procedures in your allergic patients may not be wholly scientific need not interfere with your result. What do you, or the patient for that matter, care whether the scientific explanation is satisfactory, if the treatment works? Almost anything can be good medicine and good practice of medicine if it does not harm. This is the important thing. How many procedures in your field of E.E.N.T. meet either criteria, and particularly the latter one—do no harm?

As an illustration of this in another field of surgery, Allbutt,

in 1884, in his lecture on visceral neurosis made the following statement: "A neuralgic woman seems thus to be peculiarly unfortunate. However and repeated may be her visceral neuralgias, she is either told she is hysterical or that it is all uterus. In the first place she is comparatively fortunate, for she is only slighted; in the second case she is entangled in the net of the gynecologist who finds her uterus, like her nose, is a little on one side. Or again like that organ, is running a little, or is as flabby as her biceps, so that the unhappy viscus is impaled upon a stem, or perched upon a prop, or is painted with carbolic acid every week in the year except during the long vacation when the gynecologist is grouse shooting. Her mind thus fastened to a more or less nasty mystery becomes newly apprehensive and physically introspective and the morbid chains are riveted more strongly than ever. Arraign the uterus and you fix in the woman the arrow of hypochondria. It may be for life." As a complete aside, I am always delighted but somewhat jealous when reading articles written by our British or commonwealth colleagues. Witness this short opening paragraph from a recent article: "It makes an unpromising start to confess, as I must, that this clinical study hinges on a patient whom I never met. In fact, I was on my sick bed when her impact was foreshadowed and there I had to lie, in another part of the hospital, when she made her passage through the psychiatric department."

How else might you benefit from taking a closer look at yourself? An ophthalmologist told an elderly, somewhat poor risk, cataract patient the following: "If you make the slightest move during the operation I can't be responsible for the outcome. This is a difficult operation. You must hold 100 per cent still." Very likely the surgeon, aware of the poor prognosis, made this statement so that he would not feel responsible (and guilty) if the operation was unsuccessful. I do not intend to impugn your motives or question your honesty; I only ask that you realize that it is necessary to look behind the obvious and to be aware of the various mental mechanisms that operate in all human beings.

Dr. Levine[7] says that "patients are not members of a separate species but rather are slightly varied examples of human

beings. And, second, patients are those men, women and children who, because of a hope of a cure or the lessening of their stress, are more willing than others to reveal the truth of their secret lives and the essence of their humanity. Hence, the intensive and extensive study of patients has led to a greater understanding of men and women in general."

In observing and studying yourself, not only in the context of your surgical practice but in your home and social life as well, you may benefit in various ways, not only in the practice of medicine: at times by finding some unknown and neglected item of mental hygiene and enlarging your range of opportunities for action within yourself or your patient. You may uncover some self-defeating trends or paradoxical personality needs which lead to frustration and lack of satisfaction. You may find yourself rushing like "heck" but the reasons may not be those that you think. It may be that you are running away from yourself. The next time a patient broaches a topic on which you find yourself about to reassure him or interrupt him, since you note his anxiety, stop yourself. Let him go on. Could it be that this subject is anxiety provoking to you? If so, you should know it. Anxiety is very helpful in that it is the motivating force that compels the patient to talk or to behave in such fashion and in so doing gives you much information about his problem; likewise, the absence of anxiety in a patient, when discussing some aspect of himself or some topic that logically should be anxiety provoking, may give you important insight into the patient and his attitude toward his illness. For example, the presence or absence of anxiety in a patient preoperatively is not, *per se*, necessarily helpful in your prediction of what will happen during surgery. Consider the presence or absence of anxiety in the context of the entire situation. By the term *entire situation* I mean not only the personality of the patient, the doctor-patient relationship, the attitudes or beliefs of both physician and patient, but also the relationship of the patient's family to the illness, its effect on the family, and the possible advantages or disadvantages derived by the patient from the illness. For example, if a patient has an ulcer, it vastly affects the whole family, and in a real sense, it is the family's ulcer.

Identification is one of the primary mental mechanisms that often get a surgeon in trouble. For instance, one of his patients may be an individual who is a so-called pillar of the community.[5] Failure to detect physical illness is upsetting to the doctor, himself a pillar, because serious questioning or attempts to discover emotional or psychological problems, the doctor thinks, might be considered an insult to the patient, since the doctor himself would be insulted by such an implication or approach. Doctors often do not trust themselves to deal with a problem when it seems to be clearly an emotional one or when emotional factors are paramount. Parenthetically, it is interesting that many physicians are very good at handling emotional problems in an intuitive fashion or for empirical reasons, yet, when they begin to analyze why, they get a kind of inferiority complex and often are not nearly so successful thereafter in controlling such problems. Somewhat analogous is the story about the thousand-legged worm who was asked how he controlled all those legs. At this point he couldn't. The patient with his problems and difficulties may activate similar difficulties, or at least difficulties in the same sphere, in the physician, forcing him to defensive maneuvers to avoid the anxiety engendered in himself. These maneuvers vary but, at the least, may involve changing the area of inquiry or the nature of the relationship. It is conceivable that the changes necessitated by the physician's own psychological reactions might render the treatment less effective for the patient than it otherwise might have been.

Let me reiterate in this way: It is obvious that both the doctor and the patient bring to the situation their own individual personalities. In addition, the doctor possesses superior knowledge and experience in the medical aspects of the situation. It should be pointed out that this meeting takes place in a certain cultural setting and that each individual is a product of his culture and responds as such. Most patients go to a physician with the idea that he will find some illness or abnormality that he will in turn do something about, probably by the use of drugs or other specific treatment; furthermore, the patients feel that they have had very little to

do with becoming ill and will likewise have little to do with getting well. That is, the doctor is viewed in an omnipotent role, and the patient usually longs for the doctor to live up to this role. The physician's own psychological make-up will determine what his reaction to this role will be.

Briefly, in the three preceding sections of this paper on the psychological management of the surgical patient, I have said to you that:

1. Experience has proven that psychological factors can and do enter into the genesis, development, and recovery of virtually all illnesses heretofore thought to be of an organic nature. The surgeon, like any other physician who concerns himself with only the physical factors of his patient's illness, deprives himself of much important information about the illness and deprives the patient of an essential ingredient of all types of treatment—namely, the psychological factors.

2. The personality and beliefs of the surgeon can and do influence the results he will get from certain treatment. The doctor-patient relationship, controlled in part by the physician's personal beliefs, sometimes determines the outcome of the treatment.

3. The surgeon, like all other physicians, needs to know himself as a person and as a physician, for his own mental and emotional mechanisms, his own anxieties, frustrations, identifications, and means of escape strongly influence his own life and that of all patients whom he treats.

To these ideas, which are so basic and well-known that some would call them truisms, you may respond with: "Why, yes, I believe what you have said is true. I have always believed that; but so what?" Actually, my talk could well have been entitled "That doesn't apply to me." —If on the other hand, you do accept these concepts personally and find that some parts of this paper do apply to you as individuals and as surgeons, the application of these principles will be of immense value to you.

CONCLUSIONS—OR—WHY, HOW AND WHEN PSYCHIATRY.

For your purpose, think of psychiatry from two separate aspects. In medical education, psychiatry courses give a general idea of the specialty of psychiatry, that is, when to call in a psychiatrist and what the provinces of psychiatry are. Equally important is the responsibility of psychiatry to present skills, insights, and psychological principles available from the subject and task of psychiatry itself.

Up to this time, psychiatry's association with surgery has not been the fruitful association so often true with internal medicine. For example, the literature on the subject is not very extensive, relatively speaking. There is, however, some work that I would like to call to your attention. I think that anybody remotely interested should read the various articles by Titchener, et al., at Cincinnati. They have studied the interaction of emotions and personality with surgical illnesses, treatment and convalescence. They have done this in a scientific fashion and some of the results are most interesting. In a psychiatric survey of 200 randomly selected and representative surgical patients admitted during one year to a general hospital, there was an incidence of 22 per cent of psychoses.[9] The investigators present other findings and suggestions for interpreting the data gathered. All their papers have been published within the last five years.

The literature also includes an article by Litin[10] in the *Jour. A.M.A.* on preoperative psychiatric consultations. As reported there, the psychiatrist's special skills contributed in a variety of ways to the benefit of the patient and the surgeon. Litin pointed out that patients with a diagnosis of psychosis are often not so brittle or such poor risks as they are thought to be; in fact, in some schizophrenic patients the operation may be followed by a remission, the cause of which is obscure. He emphasizes the fact that positive criteria can be used for the psychiatric diagnosis, not just making the diagnosis by exclusion.

A recent case of my own will illustrate this. I was asked to see a 45-year-old white man with unexplained abdominal pain so severe that he was addicted to morphine. Briefly, the per-

tinent observation was that this man had made an excellent adjustment in his life in every way and seemed to be well adjusted individually up until six or eight months ago, when he began to have this abdominal pain and some personality changes. At the time I saw him he presented many of the aspects of an addicted person as well as showing a great deal of regression in his behavior. It was my opinion that psychiatric problems were not responsible for this man's pain, nor could they account for the psychological picture we saw at this time. That is, both the pain and the man's psychological state were secondary manifestations of some organic process. Exploratory operation revealed a pancreatic tumor. Among older surgeons there is an aphorism, perhaps unknown by some of their younger colleagues, that with the association of unexplained abdominal pain and sudden personality change one should suspect cancer of the pancreas. The change in the personality does not seem to be related just to pain from the cancer but other explanations are not very helpful.

By very similar means—that is, the absence of positive psychological findings—I was able to diagnose, or to suggest the diagnosis of, an islet cell adenoma of the pancreas which was proven by surgery. Sometimes a psychiatrist must exercise a great deal of effort in holding to his opinion. There is nobody so tenacious as an organically minded physician who, having looked long and hard for organic illness and being unable to find it, becomes convinced that the problem is psychiatric. One such case I saw recently had had numerous tests and even an exploratory operation because of peculiar, unusual abdominal complaints. He developed a severe depression which responded to some of the newer energizers, but he just did *not* fit the picture of a psychosomatic problem, and his physician was so informed. Nothing was done until he developed additional complaints, at which time an abdominal cancer was found. These developments were certainly no reflection on his physician, for I had made no attempt to diagnose his illness other than to rule out psychiatric problems. Obviously, there are many cases in which this method of diagnosis could not be applied, *viz.*, those patients who have coexisting psychiatric and physiological symptoms.

In ophthalmological surgery, the field in which some of you practice, psychiatric research from a number of different projects and sources, has converged to help explain an observation that you made a long time ago concerning the postoperative psychoses seen with cataract surgery, which in the past has been followed by psychoses in as many as 7 per cent of the cases. With improvement of surgical technique the incidence has been reduced tremendously, and it is possible to decrease this even further. Ziskind[11] has written a very nice article pulling together the various scientific studies on the result of different kinds of isolation stress in human beings and the relationship of this stress to conditions in which the major sensory systems were interfered with, such as the eye and the ear. Greater understanding of so-called sensory deprivation or sensory patterning, as studied by D. O. Hebb, J. C. Lilly, and J. T. Shurley, has been very helpful in elucidating certain situations encountered in eye surgery particularly.

One of the first consultations that I had with a member of this audience was a telephone conversation about a postoperative patient of his. I am not certain about all the details, but the case involved cataract surgery in an elderly individual whose hearing was impaired and who developed, postoperatively, disorientation, confusion and other signs of an organic psychosis. I think the physician thought me a bit flippant, or worse, when my advice was: *a.* to cut down on sedatives and/or tranquilizers (the central nervous system already being depressed from the anesthesia and further chemical depression therefore adding to the total problem); *b.* to turn the lights on in the room and unbandage the eye as soon as possible, if that had not already been done; *c.* to install a radio in the room and leave it on at all times; *d.* to have someone from the family, someone he knew, there to talk to him. In other words, to give him as much sensory stimulation of various sorts as possible. I believe that the man was reoriented in a few hours and remained so. I have no doubt, because I have seen it in other similar cases, that this disorientation and confusion were directly the result of factors that I have suggested. The attending physician was well aware of the importance of unbandaging the eye, but I think

his case represented an accumulation of sensory deprivation or isolation in a central nervous system already depressed, and that the patient lost contact for these reasons.

At this point I would like to repeat my statement that in many cases the psychiatrist is not only unnecessary, but the surgeon will be able to do a better job than could the psychiatrist. As previously stated, bringing in psychiatry is often thought by the patient to be an insult, a threat, or worse. The surgeon has much of the family information and other data, and this, combined with the already established rapport of the primary doctor-patient relationship, is often all that is necessary. It also keeps the physician from *passing the buck*, so to speak. The comment I keep hearing is that psychiatric consultations take just too much time. I would be the first to admit that psychiatric diagnosis and treatment most often is a time-consuming affair; however, such specialized treatment is often not necessary and there are some short-cuts that will help you in many ways. Winn,[12] in a recent issue of the *Jour. A.M.A.*, presents some principles for a brief psychiatric evaluation by the surgeon. You can determine: *a.* what meaning the disease has to the patient and particularly the anxiety associated with the illness; *b.* how the patient's symptoms alter his life; *c.* whether the patient has adjusted to the reality of the therapeutic situation (Is this a defense mechanism and what are his defense mechanisms?). If this sounds difficult, I had better repeat it, because it can be approached on common-sense grounds: *a.* you should arrive at the meaning of the disease or the illness to the individual; *b.* you need to know the result of the patient's symptoms in relation to his life, or how they have influenced and will influence his life; *c.* and finally, you need to consider how he has adjusted to the illness and to the treatment proposed—whether he is too calm about something that is very serious or whether the reverse is true.

The need to view the entire situation was noted in 1883 by Oliver Wendell Holmes, who gave voice to some of this concern by saying, "I have often wished that disease could be hunted by its professional antagonists in couples—a doctor

and a doctor's quick-witted wife. Many a suicide would have been prevented if the doctor's wife had visited the victim the day before it happened. She would have seen in the merchant's face his impending bankruptcy, while her stupid husband was prescribing for his dyspepsia and endorsing his note. She would recognize the love-lorn maiden by an ill-adjusted ribbon—a line in the features— a droop in the attitude—a tone of voice—which means nothing to him . . . ."

From all sides we are hearing more about the whole person and about treating not just the disease but the patient, etc. This is always good advice. Likewise there is something to be gained from the writings of Dale Carnegie on "How to Win Friends and Influence People." It is also important to think positively and to appreciate the power of positive thinking. All this is sometimes good advice. I hope, however, that if I have done nothing else in this essay, I have avoided all of this kind of advice, which embodies rather shallow philosophies, oversimplifying the complexities involved in the living of a satisfying, productive life in spite of perhaps many obstacles. My objection is that these erstwhile panaceas constitute part of the block that prevents good psychological principles from being utilized by surgeons, and others, or from influencing the patient. Both of them, patient and surgeon, think their acceptance of these popular concepts indicates that they are psychologically sophisticated and that this is what is necessary. If reason, logic, good manners, lots of attention, rest and vacation, reduction of environmental tension, and the like, don't help, then there just isn't anything wrong with the patient, and there is some kind of irresponsibility or lack of cooperation on his part—or this is simply a bad patient. We owe more to our patient than an attentive ear and good advice. Ours must be a discerning ear; and we must be capable of understanding and explaining after we have listened. Above all, we must be capable of establishing a doctor-patient relationship that is a corrective and satisfying interpersonal experience for the patient.

Perhaps the biggest mistake we make is in the idea of selling the patient on the idea of living with his illness. It is one

thing to *face* an illness or disability. It is another to accept that condition as a permanent handicap. And the placid acceptance I refer to implies not only lack of hope but also unwillingness to compensate for the loss of function. The only way to live with an illness is to fight it, always. The blind cannot see and the deaf cannot hear; but they have other senses with which they can compensate for their losses—with which they can *fight* their losses. That is, *if* they have spirit and hope. As Kant said, "Take hope and sleep away from man, and he is the most miserable creature on earth."

At the risk of seeming contradictory, I must emphasize that in some quarters there is a tendency to overdo the psychiatric approach. It shouldn't be necessary to remind you that attention to psychological causation should not prevent an adequate diagnosis and emphasis on physical factors. Our scientific knowledge has been too dearly won to discard. My plea is that we combine all of our knowledge in the practice of comprehensive medicine, regardless of specialty.

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2500 North State Street.

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from March 13 through March 25, 1961, under the direction of Paul H. Holinger, M.D.

Registration will be limited to 15 physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk St., Chicago 12, Ill.

MULTIPLE PRIMARY MALIGNANT TUMORS.

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and
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In 1879, Billroth[1] first described multiple primary malignancies and proposed three conditions which had to be satisfied before that diagnosis could be established:

1. Each tumor must have an independent histologic appearance.

2. Each tumor must arise in a different situation.

3. Each tumor must produce its own metastasis.

Wallace[2] felt these postulates were too strict, proposing instead the following three postulates:

1. Macroscopic and microscopic distinctness.

2. Each tumor must grow in an individual way.

3. The diagnosis is confirmed by a different nature of metastasis in each case, and the probability of one metastasizing from the other must be excluded.

Warren and Gates[3] concur with Wallace regarding the strictness of Billroth's postulates. P. J. Wooley[4] divided multiple primary malignant tumors into four groups:

1. Malignant tumors may affect the same tissue and produce identical tumors or tumors of different types.

2. Malignant tumors may affect both of a pair of organs. The tumors need not be of the same histological type.

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3. Malignant tumors may affect different regions of the same system but need not be of the same histological type.

4. Malignant tumors may present as different tumors involving different systems.

Epstein's[5] classification of multiple primary malignant tumors is as follows:

1. The tumors are multicentric if they arise in the same area.

TABLE I.

Sex Ratio and Occurrence Rate.

Date	Sex Ratio Male/Female	Total Tumors	Multiple Primary Tumors	Per Cent	Author
1931	—	950	18	0.2	Hanlon[9]
1932	1/1.6	1,078	40	3.7	Warren and Gates
1933	1/1.2	2,124	71	3.3	Hurt and Broders[10]
1936	2/1	583	46	7.8	Burke[11]
1938	2.2/1	1,411	25	1.77	Kirschbaum[12]
1939	1/1.3	2,500	113	4.52	Stalker and Phillips
1943	—	685	30	4.3	Hellendahl[13]
1944	2.1/1	2,829	194	6.8	Peller[14]
1957	—	3,096	112	9.7	Wallace
1958	—	1,514	24	1.52	Fried[15]

2. They are systemic if they arise in anatomically or functionally allied organs of the same system.

3. They are coincidental if they occur as a random and accidental association in unrelated areas.

Watson[6] demanded a stricter definition of multiple primary tumor, since he considered that the multiple primary tumor must involve different organs or systems; however, he stands as the exception to the usual thinking of today.

Most of the articles written on multiple primary tumors are based upon autopsy studies involving a great number of cancer deaths, and are certainly statistically reliable. The incidence of multiple primary tumors, on the basis of European and American data, was 1.8 per cent, and on the basis of American data, 3.9 per cent.[7] In a survey of 2,829 consecutive

autopsies on cases of cancer from 1932 to 1943 Warren and Ehrenreich[8] found 194 cases of multiple primary malignant tumors, an incidence of 6.8 per cent. This was a continuation of an earlier study (see Table I) by Warren and Gates covering the period from 1926 to 1931 in which the incidence of multiple primary tumor was 3.7 per cent. They felt that this reflected a current awareness to cancer and a greater tendency to biopsy suspicious lesions for histologic examination.

The average age of onset is apparently dependent upon the sex, there being an apparent tendency for earlier onset in women than in men, and these tumors tend to occur at approximately the same age as single tumors (see Table II).

TABLE II.

Age of Onset.

Author	Age of Onset in Men	Age of Onset in Women
Fried	61.5	50
Warren	67.7	58.2
Kirschbaum	66.0	57.2

Watson found that the interval between the diagnosis of the primary tumor and the development of a second tumor was 41 months. Stalker, et al., had a 45.1 per cent occurrence of synchronous multiple primary tumors and a 54.9 per cent occurrence of metachronous multiple primary tumors. Of the latter, 26.5 per cent had an average interval of 12.3 years. Barrett[16] reports a 3.22 year interval between the primary and the development of the second tumor. Peller reported a 3.1 year interval, and Warren and Ehrenreich found a 3.1 year interval between tumors.

As indicated in Table I, multiple primary tumors tend to be statistically more frequent in males than in females.

Hellendahl stated that the familial incidence is no higher in multiple primary tumors than in single ones; however, Stalker suggested a hereditary tendency and quoted a 26.6

per cent incidence, whereas the range in the literature is 15-30.1 per cent.

Some interesting observations on pathogenesis have been made. Hellendahl found that congenital anomalies are more frequently found in cases of multiple primary tumors. This relation to congenital anomalies raises the question of whether or not cells giving rise to cancer may in themselves be congenitally abnormal. Warren and Gates theorize a congenital predisposition or susceptibility. Watson does not believe in constitutional predisposition to multiple cancers or different systems, but he does admit that some people do have a marked predisposition to multiple tumors of the same organ. He felt that this indicated a response by different parts of similar tissues to the same stimulus but would not consider this as an indication of general predisposition. Epstein discussed a biologic accident of cancer visited on one or a group of cells versus a prepared or initiated field, perhaps of considerable extent. White proposed a predisposition on the basis of a gene for cancer inheritance, a gene for localization, plus environmental stimuli. Fried mentioned a possible endocrine basis. He raised the question of coincidence versus a susceptibility to epithelial malignant disease. Hurt and Broders, and Bugher[17] doubt the coincidence theories. Wooley suggested six possible theories of pathogenesis:

1. Are there anomalies of cell division?

2. Are there mechanical developmental anomalies or multiple foci of irritation?

3. Are there abnormal chromosomes distributed in the nuclei?

4. Are there toxins acting in two different locations?

5. Is there a complex influence of an existing tumor or neighboring cells with a predisposition for tumor growths?

6. Are there multiple foci of embryonal displaced or superfluous cells?

Hurt and Broders reported a study which was made to determine the relationship of the grade of malignancy to the

frequency of occurrence of multiple primary tumors. They concluded that if a person has one tumor, the probability of his developing another varied directly with the grade of the first malignancy. They hypothesized that the average grade of malignancy in multiple primary tumors should be higher than a like group of single tumors.

Peller presented material to support the contention that a cured cancer secured protection against the development of another cancer. This was disputed by Warren and Gates who showed the carcinoma attack rate of organs other than skin to be twice that of the expected rate. Warren and Ehrenreich calculated that multiple primary malignant tumors occurred 11 times more frequently than chance would allow. Brunschwig[18] felt that the body must have some degree of resistance to the growth and invasion of cancer cells. To support his argument he discussed various animal studies, as well as the documented cases of spontaneous regression. He agreed that any immunological mechanism is relatively feeble in most people. Bashford[19] showed that animals in which tumors had developed and then been absorbed were highly refractory to subsequent inoculations. The same was true following radium exposure. Fried stated that multiple primary malignant tumors were rare because of an immunity which developed. Green and Strauss[20] found that in animals there was no increased tendency to multiple primary tumors in affected animals over unaffected animals. Boyd[21] also maintained that one cancer afforded immunity against the development of a second. Wallace stated that of 20 patients with a primary malignancy one would later develop another primary malignant tumor. Epstein wrote that the incidence of multiple primary tumors was high when at least one of them occurred in the oral cavity. Watson admitted that some patients demonstrate a marked predisposition for multiple cancers of the same organ. He felt this indicated a response by different parts of similar tissue to the same stimulus. In experiments with animal inoculation, Bashford, et al., found that the protection following absorption of tumor material from the same species as the protected animal was greatest against subsequent inoculation of the tumor absorbed and

was less against other tumors of similar histogenesis. They found that mice which were highly protected by inoculation subsequently developed spontaneous tumors of their own.

The areas most frequently involved with multiple primary malignant tumors are the colon, pharynx, stomach, and uterus. Barrett noted a tendency for multiplicity of squamous cell cancer in the buccal mucosa. Fried found that cancer of the colon is the tumor most frequently associated with another primary tumor. Eisenstadt[22] found that tumors of the gastrointestinal and gentiourinary tracts are the most frequently associated. Warren and Ehrenreich found in 194 multiple primary cancers that 95 involved the colon, 44 the skin, and 34 the pharynx (including the buccal cavity).

Since the cases to be reported in this paper are mostly of the oral cavity let us examine some of the literature dealing with this area. Wynder[23] discussed the not infrequent occurrence of primary carcinoma in the larynx, lung, and oral cavity. Epstein felt that because of the small area of the oral cavity, serial sectioning of the intervening tissues might be required to confirm the clinical impression of distinct origin. Moertel and Foss[24] used two criteria for multicentric origin of oral cancer:

1. The tumors are stimultaneous if they are clearly separated by intervals of normal mucosa.

2. They are multiple and nonsimultaneous if the most recent lesion is clearly separated from the site of excision of the initial lesion.

Lund[25] discussed the occurrence of second primary tumors of the buccal mucosa. He considered a cancer developing in mucous membrane distant to the first primary to be a new lesion rather than a metastasis. He found the incidence of a second primary tumor in the oral cavity to be 15 times greater than that expected by chance alone. In a study of mixed tumors of the oral cavity, New and Childrey[26] found a tendency toward multiplicity.

We have encountered 14 cases of multiple primary malig-

TABLE III.

Multiple Foci in Oral Cavity.

First Cancer			Second Cancer			Third Cancer	
Type	Location	Interval	Type	Location	Interval	Type	Location
Squamous	Gingiva, upper anterior	3 yrs.	Squamous	Gingiva, lower posterior	3 yrs.	Squamous	Antrum, right
Squamous	Gingiva, left lower	8 mos.	Squamous	Jaw, right lower			
Squamous	Larynx	2 yrs.	Squamous	Tongue, anterior			
Squamous	Floor of Mouth, left	13 mos.	Squamous	Jaw, right			
Squamous	Tonsil, left	21 mos.	Squamous	Tonsil, right			
Squamous	Tonsil, left	4 mos.	Squamous	Tonsil, right	14 mos.	Squamous	Floor of Mouth, left, anterior
Squamous	Larynx and Esophagogastric Junction—simultaneous						
Squamous	Floor of Mouth, anterior and Palate—simultaneous						
Squamous	Tonsil, right	3¼ yrs.	Squamous	Floor of Mouth, left, posterior			
Squamous	Floor of Mouth, anterior	10 mos.	Squamous	Tonsillar pillar, right	16 mos.	Squamous	Floor of Mouth, right
Squamous	Tonsillar Pillar, left	4 mos.	Squamous	Tonsillar pillar, right			
Squamous	Palate, left	18 mos.	Squamous	Palate, right			
Squamous	Floor of Mouth, right, anterior	13 mos.	Squamous	Tonsillar pillar, right			

nant tumors of the oral cavity plus 12 cases of carcinoma of the oral cavity associated with tumors elsewhere in the body. In a study of 80 patients with intraoral cancer, Slaughter[27] found that 18 per cent had grossly apparent multicentric lesions. Byars and Anderson[28] reviewed 166 cases of cancer of the oral cavity and found that 12 per cent had multiple oral cancer. Wilkins and Vogler[29] found that in carcinoma of the gingiva, 21 per cent of 81 cases had multiple lesions of the oral cavity.

TABLE IV.

Cancer of Oral Cavity Associated with Cancer Elsewhere.

First Cancer		Second Cancer	
Location	Type	Location	Type
Lip	Squamous	Thyroid	Follicular Carcinoma
Tongue	Squamous	Rectum	Adenocarcinoma
Gingiva	Squamous	Uterus	Adenocarcinoma
Tongue	Squamous	Stomach	Leiomyosarcoma
Floor of Mouth	Squamous	Lung	Oat Cell Carcinoma
Tongue	Squamous	Parotid	Mucoepidermoid Carcinoma
Floor of Mouth	Squamous	Lung	Adenocarcinoma
Tonsil	Squamous	Skin	Squamous Cell Carcinoma
Tongue	Squamous	Parotid	Warthin's Tumor
Larynx	Squamous	Prostate	Adenocarcinoma
Uterus	Adenocarcinoma	Right Tonsil	Squamous
Prostate	Adenocarcinoma	Lip	Squamous

Table III analyzes the 14 cases of multiple primary malignant tumors of the oral cavity that we have collected. The average interval between tumors in our series was 17.3 months. The family history did not indicate a hereditary tendency in any of our patients. We noted no tendency to increased undifferentiation between the first tumor and those developing later. All of our cases of multicentric tumors of the oral cavity occurred in patients who had a long history of heavy alcohol intake. All of them continued to drink heavily following the treatment of their first tumor. Moertel, Wynder[30] and many others have commented on the relationship between smoking and oral cancer as well as alcohol intake and oral cancer. The evidence is convincing enough to behoove the

physician to advise patients who develop oral cancer to stop using both tobacco and alcohol.

Table IV lists our cases of cancer of the oral cavity associated with cancer elsewhere in the body.

SUMMARY.

1. Billroth first described multiple primary malignant tumors in 1879. His postulates have been found too strict by later investigators.

2. The incidence of multiple primary tumors is in the range of 4.6 per cent.

3. Multiple primary malignant tumors are more common in males.

4. It is apparent that multiple primary malignant tumors occur more frequently than chance allows.

5. Some observations on pathogenesis have been surveyed.

6. The question of immunity following the development of cancer is discussed.

7. There is a tendency for multiplicity of squamous cell carcinoma in the oral cavity.

8. The incidence of multiple primary malignant tumors is higher when at least one occurs in the oral cavity.

9. Fourteen cases of multicentric carcinomas of the oral cavity and 12 cases of cancer of the oral cavity associated with cancer elsewhere have been reported.

10. The association of tobacco and alcohol with cancer of the oral cavity has been noted.

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THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, W. Va., on April 6-8, 1961.

The guest speakers on Ophthalmology are: Dr. Irving H. Leopold, Philadelphia, Pa., and Dr. Harvey E. Thorpe, Pittsburgh, Pa.; for Otorhinolaryngology: Dr. John J. Shea, Memphis, Tenn., and Dr. F. Johnson Putney, Philadelphia, Pa.

In addition to the scientific program, arrangements have been made with Mr. Philip Salvatori of Obrig Laboratories to discuss and show techniques of contact lens fitting.

For additional information please contact the secretary, Dr. Worthy W. McKinney, 109 East Main St., Beckley, W. Va.

A NEW TECHNIQUE FOR MYRINGOPLASTIC PROCEDURES.

DIETER WENDLING, D.D.S., M.D.,

Royal Oak, Mich.

The two main problems facing the surgeon during myringoplastic procedures are:

1. The exact preparation of a bed for the free graft by denuding the tympanic membrane and part of the adjacent external auditory meatal wall of its squamous epithelial layers.

2. The proper positioning of the skin graft.

If both these conditions are met, then, in my opinion, most myringoplastic procedures yield quite satisfactory results, providing the usual medical and surgical principles concerning indication for surgery, aseptic technique, and proper follow-up are strictly observed.

The denuding of the tympanic membrane and canal wall of squamous epithelium is the most time-consuming part of the operation, if done in the conventional manner. It becomes a rather short and comparatively easy procedure if the "sanding method" is employed. This is best done with a globe-shaped fine diamond burr. These burrs have been in common use among dentists for a long time. In dentistry, of course, their main use is restricted to working on extremely hard material, namely, the enamel; however, when fitted to an angulated hand-piece, they are well suited to this procedure.

The mechanical principle involved resembles that used by the plastic surgeons in their "face-sanding" procedure. Using the diamond burr under the operating microscope, employing high speed and slight pressure, should be no problem for the surgeon familiar with temporal bone surgery. There is no tearing of the soft tissues if the instrument is used with proper care and the preparation of the bed for the graft is

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accomplished in a minimum of time. An additional advantage is the fact that S-shaped external auditory meatus whose anterior wall obstructs the anterior and antero-inferior portion of the tympanic membrane and its annular region, can easily be drilled away with the same instrument. The action of the burr also provides proper hemostasis in case any bone work should be necessary.

Squamous epithelium growing around the edge of the perforation to the mucosal surface of the tympanic membrane can also be removed by the same technique with a wide margin of safety.

The manubrium of the malleus is frequently wrapped in squamous epithelium in many cases of a long-standing defect of the pars tensa. Technically, it is no particular problem to remove this tissue from the handle of the malleus by the sanding technique; however, because of the considerable vibratory action of the burr, the possibility of acoustic trauma to the inner ear structures should be kept in mind. Further investigations concerning this subject are being carried out. From my own experience, I do not believe that the short irritation of the inner ear fluids, resulting from the action of the burr on the soft tissue structures of the tympanic membrane, is any more harmful than the conventional manipulation of these structures.

SUMMARY.

A new method of denuding the tympanic membrane and adjacent external auditory meatus from its squamous epithelial layers by use of the "sanding technique" with a diamond burr is presented.

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ETHER ANESTHESIA FOR TONSILLECTOMY STUDIES IN RESPIRATORY ADEQUACY.*

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With the impressive technical advancements that have been made in the field of thoracic and cardiovascular surgery during the past decade, there has been an increased demand for greater knowledge of biochemical measurements that might reflect aberrant respiratory physiology. When deviations in respiratory function have been demonstrated by such means, causative factors proposed have included the position of the patient during the operation, the surgical procedure itself, and the anesthetic technique or agent.[1] While the surgical and anesthetic challenge for tonsil and adenoid surgery may not approach the magnitude of that for intrathoracic procedures, the importance and gravity of tonsil surgery, if judged by the number of cases done as well as by the incidence of mortality,[2] leads one to suppose that these patients deserve closer scrutiny than they now receive in order to detect possible physiologic trespass during anesthesia.

The purpose of this study was to determine the degree to which respiratory chemistries deviate during the clinical conditions imposed by an ether insufflation anesthesia technique. Insufflation of oxygen and ether by means of a suitable mouth gag has been found, clinically, to be a highly satisfactory means by which to maintain anesthesia in children undergoing tonsil and adenoid surgery. With this technique, however,

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there is no feasible way by which the patient's respiratory efforts can be assisted. Work by Elam and Brown[3] has indicated that ether as an anesthetic agent causes a significant increase in the production of endogenous carbon dioxide. Concomitantly, the same investigators concluded that alveolar carbon dioxide accumulation was consistently found during surgical planes of ether anesthesia, unless the patient's ventilation was vigorously assisted. In view of such findings, the authors undertook a clinical study of blood pH and blood gas values to determine the extent of respiratory dysfunction that might result from the use of ether as the principal anesthetic agent for tonsil surgery without the benefit of assisted respirations.

METHOD.

A total of 20 patients was used in this study, ranging in age from four to 11 years. A combination of pentobarbital and scopolamine was given intramuscularly one hour preoperatively. All of the anesthesias were given by two physician-anesthesiologists, each of whom managed ten cases. The techniques varied only with respect to the induction phase. One of the anesthesiologists accomplished induction with open-drop Vinethene® and ether with an oxygen flow of 500 cc. per minute beneath the mask. The other anesthesiologist carried out induction by means of a cyclopropane and ether sequence administered by a to-and-fro non-absorption system (reservoir bag and mask) with a total gas flow of one liter per minute. With this technique respirations were assisted throughout induction. During the initial one to two minutes after the change-over to the insufflation technique, varying flows (200-400 cc./min.) of cyclopropane were added to the mixture of oxygen and ether.

Maintenance of anesthesia in both groups of patients was by means of oxygen and ether insufflation through an appropriate size Davis-Crow mouth gag. A Foregger Copper Kettle® was utilized to vaporize the ether. Total flow of oxygen during maintenance was between 1.4 and 2 liters. For the purpose of the study all the patients were kept in clinical Stage

III, plane 2 to 3 surgical anesthesia. A 10-degree Trendelenburg position was used during surgery.

Venous blood, arterialized according to the technique of Goldschmidt and Light,[4] was drawn at the following times: *1.* when a state of unconsciousness had been accomplished; *2.* just prior to the change-over to insufflation; *3.* on beginning the dissection of the second tonsil; *4.* at the termination of the surgical procedure.

Chemical determinations performed on the arterialized venous blood included: oxygen saturation (with a cuvette oximeter), carbon dioxide content (by a modified Van Slyke-Neil manometric technique),[5] and pH (glass electrode pH meter). Calibration of the cuvette was carried out as described by the manufacturer[6] utilizing bloods of known oxygen saturation. Such values were calculated from oxygen content (Van Slyke-Neil analysis) and hemoglobin determinations. The accuracy of the oximeter readings was checked at intervals during the experimental period and was found to be within ±2 per cent of calculated values. Measurements of pH were read only after blood samples and buffer solutions had been brought to constant temperatures and suitable adjustments had been made with the manual temperature compensation control of the pH meter.

Continuous electrocardiographic tracings were recorded on an oscilloscope screen; permanent tracings were made when indicated. Additional observations were made as to the effect of the preanesthetic medication, the general course of anesthesia, amount of blood loss during surgery, and the immediate postoperative course in the recovery room.

RESULTS.

General Clinical Course.

All of the children were awake on arrival at the operating room. During the preliminary preparations for the study, they expressed no undue concern or anxiety, and all were classified as being fully cooperative. Table I gives the patients' ages and body weights as well as their hemoglobin values and the duration of anesthesia. Airway problems dur-

ing the course of anesthesia were limited to an occasional mild laryngeal stridor early during the surgical procedure while traction was being applied to one or the other tonsils. Transient breath-holding occurred not uncommonly immediately following the change-over from the to-and-fro technique

TABLE I.

Preoperative Data and Duration of Anesthesia.

To and Fro.

Patient	Age Yrs.	Wt. lbs.	Duration Anesth. Min.	HGB. Gms.
1. S.S.	6	42	20	11.8
2. P.D.	5	34	30	11.8
3. F.H.	5	45	25	13.0
4. D.S.	7	55	25	13.5
5. D.B.	6	40	23	12.5
6. M.L.	6	45	35	13.0
7. A.P.	11	64	30	13.2
8. P.S.	6	70	28	10.0
9. L.W.	5	40	32	11.0
10. W.H.	5	54	25	12.8
Average	6.2	48.9	27.3	12.3

Open Drop.

Patient	Age Yrs.	Wt. lbs.	Duration Anesth. Min.	HGB. Gms.
11. C.P.	7	90	50	11.5
12. C.H.	6	46	40	13.0
13. S.S.	6	41	35	12.8
14. C.B.	6	44	35	13.0
15. E.S.	9	80	30	12.0
16. F.S.	7	50	25	10.8
17. A.S.	5	44	40	11.8
18. B.H.	4	41	40	11.8
19. J.F.	5	46	33	12.6
20. M.P.	5	49	32	13.8
Average	6.0	53.1	36.0	12.3

to insufflation, but in each case this was remedied by adding cyclopropane to the oxygen-ether mixture for two to three minutes. Blood loss was considered to have been minimal to average in all cases except one (C.P.), in whom difficulty with oozing was not only marked during the procedure, but also continued into the postoperative period, at which time the child was transfused with one unit of blood.

Blood Chemistries During the Anesthesia.

We note from Table II that oxygen saturations were in excess of 93 per cent during maintenance of anesthesia (insufflation) with one exception, (C.P.), whose saturation value fell to 76 per cent at the time of the commencement of dissection of the second tonsil. As would be expected, the to-and-

TABLE II.

Arterialized Blood Oxygen Saturation (Per Cent).

To and Fro.

	Patient	a	b	c	d
1.	S.S.	98.5	99.5	99.5	99.5
2.	P.D.	100.0	100.0	100.0	99.5
3.	F.H.	95.5	98.5	100.0	99.5
4.	D.S.	93.0	98.5	98.5	98.0
5.	D.B.	94.0	99.5	99.5	99.0
6.	M.L.	96.5	98.0	94.5	97.0
7.	A.P.	98.0	100.0	99.5	98.0
8.	P.S.	95.5	100.0	99.5	99.0
9.	L.W.	96.5	98.5	99.0	98.5
10.	W.H.	99.0	99.0	99.5	99.5
Average		96.7	99.2	99.0	98.4

Open Drop.

	Patient	a	b	c	d
11.	C.P.	88.0	80.0	76.0	91.5
12.	C.H.	96.5	98.5	98.5	98.5
13.	S.S.	81.5	98.5	99.5	98.5
14.	C.B.	89.5	88.0	93.5	96.5
15.	E.S.	91.0	98.0	97.5	98.5
16.	F.S.	94.5	98.5	97.0	96.5
17.	A.S.	95.0	90.0	97.5	97.5
18.	B.H.	93.0	96.5	96.5	98.5
19.	J.F.	93.5	96.0	96.0	95.0
20.	M.P.	83.0	99.5	97.0	—
Average		90.6	94.4	95.0	96.8

a.—Induction.
b.—Changing from to and fro to insufflation.
c.—Beginning dissection of second tonsil.
d.—Termination of surgical procedure.

fro assisted respiration technique of induction resulted in better oxygenation during this phase of anesthesia than did the open-drop technique. With respect to carbon dioxide contents (see Table III), blood samples drawn during the open-drop to insufflation sequence gave no indication of CO_2 retention during maintenance anesthesia. The initial average

value of 46.9 Vols. per cent undoubtedly represents the effect of breath-holding just prior to loss of consciousness. The closed to-and-fro induction technique brought about an average increase in the carbon dioxide content of 5 Vols. per cent over the initial reading. This accumulated carbon dioxide

TABLE III.

Arterialized Blood CO_2 Content (Vol.%).

To and Fro.

Patient	a	b	c	d
1. S.S.	41.3	46.1	40.7	40.4
2. P.D.	36.7	46.8	39.5	41.5
3. F.H.	43.2	47.6	40.0	37.6
4. D.S.	44.3	49.1	43.1	42.6
5. D.B.	45.8	49.0	46.1	43.4
6. M.L.	35.6	43.2	40.0	34.0
7. A.P.	47.2	50.5	44.4	44.4
8. P.S.	41.0	44.0	42.0	39.5
9. L.W.	39.3	45.2	38.8	39.0
10. W.H.	41.4	44.5	41.2	39.5
Average	41.6	46.6	41.6	40.2

Open Drop.

Patient	a	b	c	d
11. C.P.	44.6	40.3	38.7	39.6
12. C.H.	47.5	42.2	39.2	39.8
13. S.S.	49.7	42.1	41.7	41.7
14. C.B.	48.4	48.6	45.7	43.5
15. E.S.	48.4	42.4	42.1	43.1
16. F.S.	52.3	44.1	42.8	42.0
17. A.S.	47.5	44.6	40.3	40.6
18. B.H.	42.8	41.3	41.4	37.5
19. J.F.	39.3	34.2	35.0	39.3
20. M.P.	48.6	42.3	41.7	—
Average	46.9	42.2	40.9	40.8

a.—Induction.
b.—Changing from to and fro to insufflation.
c.—Beginning dissection of second tonsil.
d.—Termination of surgical procedure.

was completely eliminated by the time the first tonsil had been resected. pH determinations (see Table IV) showed no remarkable variations in acid-base balance, except for a mild respiratory acidosis during induction with the to-and-fro technique. With the change-over to insufflation, the average pH returned to a presumably normal value.

TABLE IV.

Arterialized Blood pH.
To and Fro.

	Patient	a	b	c	d
1.	S.S.	7.41	7.38	7.43	7.44
2.	P.D.	7.40	7.37	7.41	7.38
3.	F.H.	7.46	7.42	7.42	—
4.	D.S.	7.35	7.30	7.38	7.41
5.	D.B.	7.36	7.34	7.40	7.44
6.	M.L.	7.38	7.35	7.38	7.40
7.	A.P.	7.38	7.34	7.42	7.44
8.	P.S.	7.30	7.38	7.41	7.49
9.	L.W.	7.40	7.35	7.48	7.48
10.	W.H.	7.41	7.37	7.45	7.47
Average		7.39	7.35	7.42	7.44

Open Drop.

	Patient	a	b	c	d
11.	C.P.	7.38	7.38	7.40	7.40
12.	C.H.	7.36	7.38	7.38	7.38
13.	S.S.	7.38	7.40	7.41	7.40
14.	C.B.	7.43	7.42	7.45	7.48
15.	E.S.	7.42	7.46	—	—
16.	F.S.	7.40	7.48	7.50	7.50
17.	A.S.	7.36	7.41	7.43	7.40
18.	B.H.	7.42	7.35	7.33	7.38
19.	J.F.	7.40	7.45	7.42	7.40
20.	M.P.	7.45	7.49	7.47	—
Average		7.40	7.42	7.42	7.42

a.—Induction.
b.—Changing from to and fro to insufflation.
c.—Beginning dissection of second tonsil.
d.—Termination of surgical procedure.

Electrocardiographic Changes.

Aside from minimal changes in cardiac rate, 18 of the children showed no variation in their electrocardiographic tracings. The remaining two patients developed conduction defects during the latter part of the maintenance phase of anesthesia. This consisted of a nodal rhythm in one child (J.F.), and a first-degree heart block in the other (C.B.). Both of these abnormal patterns returned to normal sinus rhythms before the termination of the procedures.

DISCUSSION.

Whitteridge[7] voiced the classical description of the influence

of ether on respiratory behavior in stating that the respiratory center was stimulated reflexly by ether until the deeper planes of surgical anesthesia were reached, at which time there was a reduction in both tidal volume and minute volume. In a recently edited textbook of pharmacology,[8] the sectional author states that body metabolism is reduced by ether, and, in the absence of anoxia or carbon dioxide retention, there is no upset in acid-base balance.

In an excellent review of pediatric anesthesia, Stephen[9] defined good anesthesia as that which caused "minimal disturbance of the patient's basic physiologic functions." Included in this review was a critical analysis of the open-drop technique in which Stephen pointed out that the technique was essentially limited to ether, which produces metabolic acidosis when used in relaxing concentrations. Watrous[10] stressed the importance of actively supporting respirations during all types of inhalational anesthesia so as to combat the tendency of such agents to cause hypoxia and respiratory acidosis. Beecher[1] has presented data to show that patients receiving ether for thoracic surgery develop a combined respiratory and metabolic acidosis unless the patient's ventilatory efforts are vigorously assisted. He called attention to the fact that the oxygen tensions were generally satisfactory at the time when the carbon dioxide tensions were dangerously high. Similar studies[11,12] substantiated this noteworthy clinical report and provided further evidence as to the possible disturbance in acid-base balance resulting from ether anesthesia. Bunker[13] found that most infants and about 50 per cent of children between the ages of one to 12 years develop moderate degrees of metabolic acidosis during ether anesthesia.

In contrast to patients undergoing thoracic procedures, Beecher[14] was able to show that adults anesthetized with ether, but kept in a supine position, failed to develop metabolic acidosis; furthermore, they have no tendency to accumulate alveolar carbon dioxide or build up fixed acids. In extrathoracic procedures under deep ether anesthesia, Gabbard[12] noted that alveolar ventilation was sufficient to maintain normal arterial carbon dioxide tensions. In several instances,

the ventilation was actually increased to the point of causing respiratory alkalosis.

In the present study, there are numerous clinical factors which might possibly lead to findings of respiratory inadequacy. These are:

A. Depressed minute volume due to deep surgical planes of anesthesia.

B. Unsupported respirations.

C. Trendelenburg position, in which diaphragmatic efficiency is impaired.

D. The age of the patients.

E. Surgical manipulation, retraction and packing in the immediate vicinity of the glottis.

The composition and analysis of blood chemistries in our series of patients failed to show that the above factors materially affected respiratory function. With the exception of one patient (C.P.) in the open-drop induction group, all the children showed oxygen saturations of 93 per cent or better throughout the maintenance period (after change-over to insufflation). We have no explanation for the exceptionally low oxygen readings of this one patient who demonstrated no unusual airway problem. Of some significance perhaps is the fact that this child had a chronic upper respiratory-tract infection and a rectal temperature of 100° F. During the procedure, the blood was considered to be dark by both the anesthesiologist and the surgeon. This same patient had excessive oozing postoperatively.

During the maintenance phase of anesthesia, the carbon dioxide and pH readings showed no indication of either respiratory or metabolic acidosis. Utilizing the to-and-fro nonabsorption technique there was the expected carbon dioxide retention which was reflected in a small drop in the average arterial pH. The greatest concern over such a retention of carbon dioxide was the possibility that abnormal ventricular rhythms might occur in association with the high blood levels

of carbon dioxide during the induction phase (Lurie, et al.[15]), as well as during the period immediately following the change-over to an open insufflation system when the accumulated carbon dioxide might be rapidly eliminated (Brown and Miller[16]). Such fears failed to materialize, as all the children in the to-and-fro induction group showed normal electrocardiographic tracings throughout their courses of induction and maintenance. Reference to Table II shows that these patients were extremely well oxygenated, a factor which, according to Jacoby,[17] is most important in preventing abnormal cardiac rhythms.

It is important to point out that all of these cases were managed in conjunction with cooperative and proficient otolaryngologists, highly cognizant of airway problems. We certainly do not mean to infer that patients undergoing tonsillectomies with this method never develop laryngeal stridor, hypoxic episodes, or cardiac arrhythmias. We do believe, however, that the information presented here shows that a properly conducted insufflation technique can fulfill Stephen's criteria of good anesthesia by causing "minimal disturbance of the patient's basic physiologic function", and thus provide safe and reliable anesthesia for children undergoing such elective surgery; nevertheless, there should be no question on the part of the anesthesiologist or the surgeon that an alternative endotracheal technique and a means of aiding of supplanting the patient's exchange be immediately available in the event respirations become depressed, obstructed or otherwise ineffective.

SUMMARY.

A study was conducted to determine the presence or absence of aberrant respiratory function during ether-insufflation anesthesia for tonsil and adenoid surgery. Analysis of serial blood chemistries showed that oxygen saturation in all but one patient was in excess of 93 per cent during maintenance anesthesia, while during the same phase carbon dioxide and pH values showed no remarkable variations.

Electrocardiographic monitoring failed to show any ab-

normal rhythms during induction or maintenance anesthesia. Minor conduction changes were noted in two cases late in the course of anesthesia.

Such data have demonstrated that in children with an age range of from four to 11 years satisfactory oxygenation and acid-base balance may be maintained in deep surgical phases of ether anesthesia when administered by an insufflation technique.

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL 34TH ANNUAL SPRING CONGRESS.

The Gill Memorial Eye, Ear and Throat Hospital will hold its Thirty-Fourth Annual Spring Congress in Ophthalmology and Otolaryngology and Allied Specialties, April 3-8, 1961. There will be 20 guest speakers and 50 lectures.

CURRENT UTILIZATION OF TRACHEOTOMY AS A THERAPEUTIC MEASURE.*†

A Review of the Literature and an Analysis of 526 Cases.

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Tracheotomy has long been a fascinating operation in medical annals. Caelius Aurelianus[1] records the performance of a tracheotomy in Rome in the First Century, B.C. It has been referred to by Galen and was certainly performed in 1546 by Antonio Musa Brasavola.[2] The word "tracheotomy" appeared in the works of Thomas Fienus in 1649. The operation at that time was done for the relief of high obstruction in the airway. For this purpose, tracheotomy came to be a clearly indicated procedure and has been accepted by physicians to this day. In recent years, a changing concept of the function of tracheotomy has developed which has extended the usefulness of tracheotomy and changed many of the basic ideas which previously surrounded this operation. It is the purpose of this paper to examine the present ideas with relation to current teaching, as well as current practice in utilizing the procedure and to consider possible avenues whereby future progress may be made.

OBSTRUCTION TO THE AIRWAY AS AN INDICATION FOR TRACHEOTOMY.

Although the classical indication for tracheotomy has been high obstruction in the airway, it is noteworthy that today many authors tend to create a single category without defining the variations in high obstruction which drastically alter one's clinical judgment with regard to management. In high respiratory obstruction, a distinct difference exists be-

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tween supraglottic lesions and lesions involving the cartilaginous box of the larynx.[3] These deserve particular separation in teaching, so that an improvement in practice may occur.

Eve[4] has pointed out how obstruction to the airway may occur when the epiglottis is inadvertently forced into the airway in such a manner that it cannot be displaced. He examined a victim of electrocution. The epiglottis was firmly wedged in the superior laryngeal aperture by the muscular forces generated by the current. Eve felt that if the electrical current had not killed the patient, he would have been asphyxiated. He further suggests that certain sudden deaths observed during bombing attacks may be due to the force of the shock wave of the explosion jamming the epiglottis into the superior laryngeal aperture at a time when the patient was in the phase of expiration. Under these circumstances, having insufficient air in the lung to force the epiglottis out of the superior laryngeal aperture, the patient is thereby asphyxiated. He suggested this as an explanation for the observed fact that two persons in a blast area may be standing side by side uninjured by any flying particles. One lives, and the other dies. In Eve's opinion, the one who dies may have been at the end of an expiratory cycle when the shock wave struck and was, therefore, unable to force the occluding epiglottis out of the superior laryngeal aperture.

A similar situation has been described by Miller[5,6] with regard to acute epiglottitis in children. One of the characteristic features of the dyspnea in acute epiglottitis is its variability. This is due to the fact that swollen and edematous tissues of the epiglottis and superior laryngeal aperture are not fixed by the laryngeal cartilages. These tissues produce obstruction which varies with the degree of swelling, the position of the head, the contraction of the pharyngeal musculature, and the force of respiration. With a swollen mass of tissue overlying the superior laryngeal aperture, it is easy to understand how a situation such as Eve has suggested could occur. The patient suffers a bout of paroxysmal coughing, exhausting air in the lung, and then attempts to inspire quickly, pulling the swollen tissues of the supraglottic area into

the glottis like a cork into a bottle. The patient with insufficient air in the lung is unable to expel the edematous tissues from the airway and is instantly suffocated.

The entire supraglottic airway is subject in greater or less degree to this same sort of variable obstruction. This type of obstruction should be recognized as a clinical entity distinctly different from obstruction in the region of the cartilaginous box, where the size of the airway tends to diminish by regular progression. Glottic and subglottic obstruction involves this cartilaginous box and produces a clinical picture where a clear-cut relationship exists between the symptoms of obstruction and the need for tracheotomy. It has been our practice to separate sharply those obstructive lesions arising in the supraglottic area where obstruction is variable and the moment of asphyxia is completely unpredictable. For these, only three possible courses can be observed: *1.* relieve the obstruction; *2.* literally stand by the patient prepared to do an immediate tracheotomy, or *3.* tracheotomize in order to guarantee a free airway until such time as the supraglottic obstruction is relieved. With regard to obstruction at the level of the glottis, it is possible to allow an obstruction to exist until that point is reached where the patient shows, in addition to stridor and retraction, the evidence of inadequate ventilation as demonstrated by pallor, restlessness, fatigue, sweating, and the inability to sleep.

The concept of tracheotomy as a method of relieving high obstruction exclusively persisted to modern times. As late as 1934, Figi[7] could report 200 consecutive patients on whom 206 tracheotomies were performed. In this group, 137 were done for carcinoma of the larynx, 25 for goiter, 14 for foreign body, and eight for laryngeal papillomata. In none of the cases reported by Figi was there any mention of tracheotomy for the relief of bronchial obstruction.

THE ROLE OF TRACHEOTOMY IN THE MANAGEMENT OF PULMONARY AND VENTILATORY PROBLEMS.

The remarkable extension of tracheotomy to the treatment of pulmonary and ventilatory problems has been largely a

contribution of the otolaryngologist. As early as 1911, Chevalier Jackson[8] called attention to the fact that a patient may literally drown in his own secretion; but it remained for Lee, Tucker, and Clerf[9] to demonstrate by bronchoscopy that atelectasis was caused by the mechanical obstruction of bronchi by secretion and that removal of the obstruction restored pulmonary ventilation. This concept was also aided by the contributions of Coryllos.[10] The acceptance of this idea by the profession generally was slow, but in hospitals with well established bronchoscopic services, postoperative atelectasis was frequently managed by bronchoscopic aspiration. Where the conditions that produced the accumulation of secretion were non-recurring, bronchoscopy proved to be the ideal method of treatment and remains so today. The success of bronchoscopy was less satisfactory where failure of the expulsive mechanism of the lung permitted the re-accumulation of secretion. The work of Galloway[11,12,13,14,15] demonstrated the importance of airway obstruction by secretion in poliomyelitis, and the usefulness of tracheotomy in overcoming this constantly recurring obstruction. The accuracy of his observations was rapidly attested by numerous observers,[16,17,18,19,20,21,22,23,24] so that tracheotomy in bulbar poliomyelitis became an accepted method in this country. In England, it was not until Lassen's[25] report of the Copenhagen epidemic of poliomyelitis in 1953 that the use of tracheotomy for secretory obstruction was widely accepted.[26]

Tracheotomy came to be applied to a great many different disease situations, including tetanus,[27,28,29] botulism,[30] barbiturate poisoning,[31] eclampsia,[32,33,34] leprosy,[35] hydrophobia,[29] and certain allergic emergencies.[36] In our own clinic we were able to demonstrate marked improvement in the management of neurosurgical patients as was reported by Taylor and Austin.[37] Tracheotomy has come to be accepted as an essential part of the management of patients with depression of the central nervous system related to neurosurgery,[38] acute head trauma,[39,40,41] and other causes of unconsciousness.

Kime[42] has recommended that the depth of unconsciousness be related to levels of anesthesia as a criterion for election of

tracheotomy. A unique application of tracheotomy in neurosurgical problems is its use by Jackson and Carter[43] to prevent the building up of air pressures in the upper respiratory tract, in order to prevent the escape of air into the meninges from a skull fracture communicating with the upper respiratory tract.

The acceptance of tracheotomy in general surgical services was somewhat slow, though everyone recognized the necessity of tracheotomy in thyroid surgery[44] where the indication was usually high obstructive dyspnea. The demonstration by Haight[45] in 1938 that the introduction of an aspirating catheter into the tracheobronchial tree would reduce postoperative pulmonary complications by mechanically removing secretions and stimulating cough to fortify the expulsive mechanism of the lung showed the usefulness of this concept in general surgery.[46,47,48]

In thoracic surgery, tracheotomy has become an important part of the armamentarium for a number of reasons.[49,50,51] Where there has been a pulmonary resection and a marginal ventilating area remains, the accumulation of secretion is particularly intolerable to the patient. A similar interference with respiration is encountered in crushing injuries of the chest.

D'Abreu[52] in his recent text states that tracheotomy should be performed without hesitation as early as possible in crush injuries of the chest whenever paradoxical movements, ineffective respiration, dyspnea, cyanosis, and the inability of the patient to cough up bronchial secretion are obvious or are developing.

The treatment of burns constitutes a peculiar indication for tracheotomy because a variety of elements enter into the indications. Burns of the neck may be associated with edema of the burn area. If the face is involved, the eyes and mouth may become closed by swelling; and when vomiting occurs, there is danger of aspiration. An extension of this edema to the deeper tissues may interfere with respiration and cough. A further factor is the possibility of injury by the inhalation

of hot or irritating fumes. Moncrief[53] feels that air can be cooled so rapidly that burns from this cause are rare, but notes that steam and irritant smokes can produce thermal or caustic injury to the tracheobronchial tree itself. Under these circumstances, we have seen serious damage to the respiratory tract with a threat to the airway from edema, exudate, and slough of the mucosa with long term serious impairment to pulmonary function. Of a thousand burn cases observed by Moncrief, 84 required tracheotomy.

TRACHEOTOMY IN RESPIRATORY FAILURE.

The current interest in pulmonary function has encouraged observers to relate respiratory problems to the total function of respiration. In addition to the local problem occurring in the lung, the failure of sufficient central nervous system drive to maintain adequate ventilation and the effects of such loss must be considered.[54,55,56,57,58] Among the earliest attempts to maintain ventilation under these circumstances, the Drinker Respirator was a definite advance. It soon became apparent, however, that accumulation of secretion could nullify the usefulness of this type of treatment. Tracheotomy, therefore, was employed to permit access to the airway. The use of this cumbersome apparatus which competed with the tracheotomy tube for the neck area complicated the treatment but constituted a distinct advance.

Another solution to the problem of loss of respiratory drive was the use of a cuffed oro-tracheal tube with forced ventilation through the mouth. Such tubes are quite effective for brief periods of time, but if it is necessary to maintain ventilation in this way, several disadvantages appear. They are not well tolerated by a conscious patient, they render aspiration of bronchial secretion difficult, they frequently lose lumen by encrustation of secretion within the tube itself, and they tend to increase the amount of dead space. Respiratory failure is encountered not only in disease of the central nervous system and muscles, but also in emphysema and acute respiratory infections. Westlake[59] studied the arterial blood gases of 30 patients with acute respiratory infection. In six of these who

had no previous chest difficulties, the arterial carbon dioxide pressure was normal; however, 18 of 24 with a history of chronic bronchitis and emphysema disclosed an arterial carbon dioxide tension five to 32 millimeters above that obtained on recovery. He considered these to be in acute respiratory failure. The accumulation of carbon dioxide in sufficient concentration produces a profound narcotic effect on respiratory drive, so that functionally there is, in addition to hypercapnia, increasing anoxemia and respiratory acidosis. Westlake enumerates the following symptoms: increased intracranial pressure, choked discs, headache, mental confusion, hallucinations, sweating, myoclonic jerking, increased cardiac output, pulmonary hypertension, and congestive heart failure. In patients such as these, the administration of oxygen reduces the anoxic drive and thereby diminishes ventilation with the production of a vicious circle consisting of increasing hypercapnia and increasing central nervous system narcosis. By increasing ventilation, the carbon dioxide can be removed, and respiratory function tends to be restored. In these circumstances, the use of a tracheotomy tube with a cuff surrounding it gives a sealed airway for ventilation which can be maintained satisfactorily by the use of one of the newer mechanical respirators, such as that described by Bennett. With this type of apparatus, a firm connection between the tracheotomy tube and respirator is necessary; but when an air-tight system has been achieved, the patency of the airway is easily maintained and ventilation can be satisfactorily carried on.

A tracheotomy tube devised by Dr. Thomas Cannard and the author has been found extremely satisfactory in situations where assistance to respiration is required. The fundamental ingredients of this tube are the inflatable cuff which surrounds the tube and the fitting on the inner cannula. When the tube lies in place, this cuff is in the trachea and is inflated so that a tight system is developed between the airway and the tracheotomy tube. The inner cannula of this tube is provided with a fitting that allows a tight connection to be made by the gas apparatus using the Foregger fitting. This permits the full diameter of the tracheal tube to be used for ven-

tilation and diminishes resistance within the system. It can be used for long periods of time, the cuff does not cause injury to the trachea, and it can be fitted easily to various types of ventilating apparatus which are now available (see Fig. 1).

TRACHEOTOMY IN CHRONIC PULMONARY INSUFFICIENCY.

Recent interest has been demonstrated in the treatment of chronic pulmonary disease, such as emphysema and the pul-

Fig. 1. The Cannard-Atkins tube, showing the occluding cuff inflated and the inner cannula with the Foregger type fitting for connection to a mechanical ventilating device. (Made by George P. Pilling & Son Co.)

monary aspects of cystic fibrosis by a technique called tracheal fenestration.[60,61,62,63] This is a surgical procedure that results in a firmly epithelialized mucocutaneous opening into the trachea provided with lip-like valves that can act as a closing mechanism. It is intentionally designed to obviate the wearing of a metal cannula. The opening thereby produced in the trachea admits semi-rigid catheters for the purpose of aspi-

rating bronchial secretion from these patients with chronic pulmonary disease. The advocates of the method contend that the critical factor in the disability of such disease is not the quantity of secretion but the reduced pulmonary reserve associated with a failure of the cough mechanism. They claim the effects of aspiration extend beyond the immediate relief from discomfort and asphyxia. Tracheal aspiration is often followed by prolonged improvement so that the frequency of aspiration becomes less and less, and exercise tolerance improves. This would parallel the experience one sees in patients wearing conventional tracheotomy tubes, where the improvement is attributed to the elimination of the effects of infection which almost automatically follows an improvement in pulmonary drainage. Following the fenestration operation, patients aspirate themselves and learn to control the flaps with digital pressure, so that cough and expectoration via the mouth can be carried out normally. The proponents of this procedure warn against indiscriminate use and find it no short-cut therapy for profuse expectoration, cough, or pulmonary suppuration. The technique described by Rockey involves an operative procedure of some four hours' duration with a temporary tracheotomy placed about the seventh tracheal cartilage. Our personal experience with the method is insufficient to evaluate it. The idea appears to have merit, though a simpler approach to the airway would seem possible and highly desirable. A further trial of the method is justified.

TECHNICAL CONDITIONS EFFECTING SUCCESS IN TRACHEOTOMY.

Perhaps the most important single element in the successful application of the operation of tracheotomy is timing. The tracheotomy which is done too late is generally recognized as having a severely limited value.[64,65,66] This is true regardless of the indication for which the procedure is done. For this reason, prophylactic tracheotomy has been employed more and more in those situations where experience indicates that a need for tracheotomy may be reasonably expected.

Almost as important as timing, is planning. The proper respect for tracheotomy as an operative procedure with inherent hazards is frequently overlooked, particularly by the

inexperienced. A failure to appreciate that the patent airway is the thing to be acquired rather than the tracheotomy has caused physicians to make injudicious moves in unpropitious circumstances. Wherever possible, an airway should be obtained and maintained. Davis, Kretchmer, and Bryce-Smith[67] encountered complications in 54 of 155 tracheotomies. Of these 54, there were 20 major complications. Fourteen of these, or 70 per cent, occurred in tracheotomies which were done as emergencies. The acquiring of an airway is usually best accomplished by the introduction of a bronchoscope. This not only gives an airway but also permits one to examine and sometimes correct the causes of obstruction. An unrecognized foreign body or exudate may be removed with restoration of the airway, so that tracheotomy may be avoided. If a bronchoscope is not available, the use of an oro-tracheal or naso-tracheal tube will permit one to carry out ventilation and aspirate secretion. As a temporary expedient, the oral path to the airway is usually safer and faster. Once an airway has been achieved, the patient should be taken to the operating room where the tracheotomy can be done without haste with the advantage of good light, good position, good assistants, and the safeguards for emergency treatment which are normally found in an operating room. An attempt to do tracheotomy in a bed or respirator with poor light and poor assistants is not to the patient's best interest.[68] Seldom is a patient so sick that he cannot be taken to the operating room in his bed.

Of particular concern is the use of tracheotomy in small children.[69,70] It has long been our practice to stabilize the trachea and guarantee an airway by the use of an oro-tracheal airway, usually a bronchoscope which is held by an assistant and withdrawn as the tracheotomy tube is introduced. In the absence of a dependable assistant, the bronchoscope can be used as a diagnostic instrument and then removed. A fairly rigid endotracheal tube is then introduced to maintain the airway and stabilize the trachea. An anesthesiologist or nurse anesthetist can be depended on to handle such an airway expertly. This avoids the many complications which are peculiar to tracheotomy in the very young.[71]

A constantly present problem in the management of the tracheotomized patient is postoperative care.[72,73,74,75,76] This is complicated by the constantly changing personnel which exists in modern hospitals, so there is the necessity of maintaining a continuous program of education for the nursing staff.

The ideal in postoperative care of the tracheotomized patient is to maintain the airway as it would be if the normal expulsive and ventilatory forces were at work. This can usually be accomplished when the cough reflex is intact, if there is no serious underlying pulmonary disease and if there is meticulous nursing care. The problem becomes extremely difficult, however, when there is serious impairment of the cough reflex or of ventilation. Some concern has been expressed about the effect of aspiration on the circulatory status of debilitated patients by Brattstrom[77] and on the effect of rapidly lowering carbon dioxide tension following the opening of the airway in hypercapnic patients by Negus.[78] In our practice, we have never recognized any difficulty in the postoperative management of the tracheotomized patient from these causes.

EVALUATION OF CLINICAL RESULTS.

In an effort to evaluate the clinical application of tracheotomy in modern hospital practice, the tracheotomies performed in two teaching hospitals from 1954 to 1958 have been reviewed. The findings are indicated by the table prepared from the survey. It is to be noted that among the 526 cases, there was no serious or significant difference in emphasis between the practice of the two hospitals; so that the figures of one would fairly accurately reflect the practice of the other. Of the 526 cases, 13 initially had cricothyroidotomies. Among the reasons for doing the cricothyroidotomy, the most common was laryngoptosis in a very ill patient, where it was considered that the simplest possible procedure was the most appropriate for that particular moment. In all instances in which the patient survived, the tracheotomy tube was replaced in the trachea at a subsequent procedure. It is the practice in

TABLE I.

Survey of Tracheotomies—1954-1958, Inclusive.

A. Total number of tracheotomies	526
1. Cricothyroidotomies	13
2. Tracheotomies	513
B. Number done by bronchoesophagology	408
1. Associated bronchoscopies	66
2. Preliminary intubation	33
C. Number done by other services	118
D. Service primarily responsible:	
1. Neurosurgery	124
2. Neurology	30
3. General Surgery	146
4. Bronchology	65
5. Medicine	104
6. Pediatrics	21
7. Plastic Surgery	20
8. Otolaryngology	6
9. Urology	6
10. Ophthalmology	1
11. Gynecology	1
12. Radiology	2
E. Children Under 12	44
1. Children under three	34
2. Decannulated at discharge	25
3. Not decannulated at discharge	14
F. Indication:	
1. Laryngeal or above (mechanical obstruction)	
a. tumor	72
b. inflammatory	27
c. foreign body	1
d. trauma	28
e. congenital	2
2. Tracheal	38
3. Tracheobronchial (secretory obstruction)	327
4. CNS depression	
a. neurologic	155
b. metabolic	34
c. toxic	33
d. trauma	1
5. Prophylactic	99
6. Cardiac	51
G. Complication:	
1. Bleeding	1
2. Pneumothorax	2
3. Stricture	1
H. Results:	
1. Number recovered	226
a. decannulated	165
b. not decannulated	61
2. Deaths	300
a. primarily due to tracheotomy	2
b. other causes	298
3. Number autopsied	129
a. tracheitis	4
b. pneumonia	99
c. lung abscess	21
d. thermal burns to lungs	2

these hospitals that the Broncho-esophagologic Service, which is intimately associated with the Otolaryngologic Service, carries the responsibility for the management of tracheotomy problems. In 99 of the 408 cases done by the service, there was sufficient evidence of obstruction at the time of tracheotomy to require the use of an auxiliary airway during the operation. One may recognize the role which tracheotomy performs in a wide range of situations by observing the variety of services to which the patients were primarily assigned. The small figure for the Neurology Service can be attributed to the fact that for the most part cerebral vascular accidents are admitted to the medical service for care, with admission to the neurology service reserved for difficult diagnostic problems.

The long duration of cannula life which is necessary in certain children is shown by the 14 who were discharged from the hospital wearing their tracheotomy cannulas. Of 23 children tracheotomized for acute inflammatory lesions of the larynx, seven were done for supraglottic obstruction, and 16 for involvement in the glottic or subglottic area. Obstructive lesions in the trachea or above represented the indication for tracheotomy in a total of 168 cases. It is to be noted in reviewing the indications for which the tracheotomy was done that in certain cases, more than single indication was encountered.

Some progress with the idea of doing tracheotomy early is indicated by the 99 prophylactic tracheotomies represented in this group. Serious operative complications were few, but point out the need for maximum safety in the performance of tracheotomy. Results indicate the number of patients recovered and discharged from the hospital. Certain of these patients were discharged wearing their cannulas for various reasons, including the children noted above plus patients whose obstruction was not completely relieved, or patients with tumors discharged to continue X-ray therapy as outpatients. The 300 deaths in this group indicate the application of tracheotomy to the seriously ill patient. A death due to respiratory obstruction would represent loss of a patient by default.

As indicated above, the problem of maintaining a clear airway in the patient with serious impairment of ventilation and cough constitutes a real challenge in the postoperative care of these patients. Undoubtedly, many of these patients had their pulmonary complication at the time tracheotomy was done, but it is to be hoped that more effective control of inflammatory pulmonary complications can be achieved in these desperately ill patients. At the same time, it must be stressed, in connection with the autopsy findings, that the pulmonary lesions were not the primary cause of death.

COMMENT.

The above findings are in accord with the findings of Fitz-Hugh[79,80,81,82] and his colleagues, who have noted the change in the application of tracheotomy to a broad group of problems. There is some indication that this concept is not universally practiced, particularly in the non-teaching hospitals. Whitaker and Lee,[83] who noted the performance of 25 tracheotomies in a hospital performing 10,709 operations in the same period, felt that the procedure should have been more widely used. Baker and Wise[84] found that high obstruction overshadowed lower airway obstruction as an indication for tracheotomy by two to one. It would seem, therefore, that a category or syndrome should be presented to the medical profession in which these newer indications for tracheotomy are clearly outlined without being attached to any particular disease entity, so that whenever this physiologic disturbance is encountered, the needs of the situation will be clearly recognized.

The present concept of the indications for tracheotomy[26,85,86,87,88,89] is not in dispute. What seems to be necessary to develop clearly in the mind of the profession are three concepts: *1.* When tracheotomy is needed, the sooner it is done the better. *2.* Obstructive lesions of the supraglottic area are dangerous, and the degree of obstruction is inconstant; whereas, obstructions arising in the cartilaginous box of the larynx tend to be regular and predictable. *3.* Failure of the expulsive mechanism of the lung from whatever cause threatens life

and is an indication for tracheotomy. The operation offers the following advantages: *a*. It removes obstructive secretion from the airway and gives easy access to it. *b*. It discourages aspiration of pharyngeal or gastric contents. *c*. It reduces the dead air space. *d*. It permits assistance of inadequate ventilation by positive pressure breathing apparatus.

SUMMARY.

A review of the literature and analysis of the utilization of tracheotomy in 526 cases is presented.

The changing and growing scope of tracheotomy can be seen by comparing earlier reports of its use with those more recently published and with the clinical material presented.

Obstructive lesions of the upper airway should be separated clinically into supraglottic and laryngeal categories.

Obstructive lesions of the lower airway should be recognized as a single category found in many different disease situations, all characterized by a failure of the expulsive mechanism of the lung.

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3400 Spruce Street.

CORNEAL GRAFTS IN MIDDLE EAR SURGERY.

A Preliminary Report.*

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Colorado Springs, Colo.

Viable tissue has previously been used in surgery for the restoration of hearing. The tissue used for this has been skin, either as a free graft, any of the methods of sliding skin grafts,[1] vein grafts,[2,3] and mucous membrane grafts. Closure of perforations has likewise been accomplished by the use of cigarette paper, egg membrane, silver nitrate, trichloracetic acid, with or without antibiotic agents.[4] Foreign, non-viable material (gelfoam) has been used in stapedial footplate surgery.

Four essentials are necessary for the maintenance of the viability of a graft in middle ear surgery: first, the field must be free of infection; second, there must be immobility of the graft; third, there must be adequate blood supply; fourth, in tympanoplasties the graft must have free mobility in order to have an air containing middle ear cavity. To acquire the fourth demand, an epithelial and "endothelial" lined graft is best suited; otherwise the graft becomes undesirably fixed to the inner wall of the middle ear.

Corneal grafts are ideally suited for middle ear surgery. Previously, corneal transplants, both homografts and heterografts, have been largely confined to improve vision or in therapeutic grafting of the eye.

The human cornea averages 11.6 mm. in the horizontal diameter, in the adult, with the vertical diameter slightly less. It is slightly thinner by 0.8 to 0.9 mm. in the center than at the periphery where it is 1.0 to 1.1 mm. in thickness. The cornea possesses no blood vessels except at the periphery where it receives its nutrition from the capillary loops. The

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*From the Hearing Institute of the Colorado Springs Medical Center.

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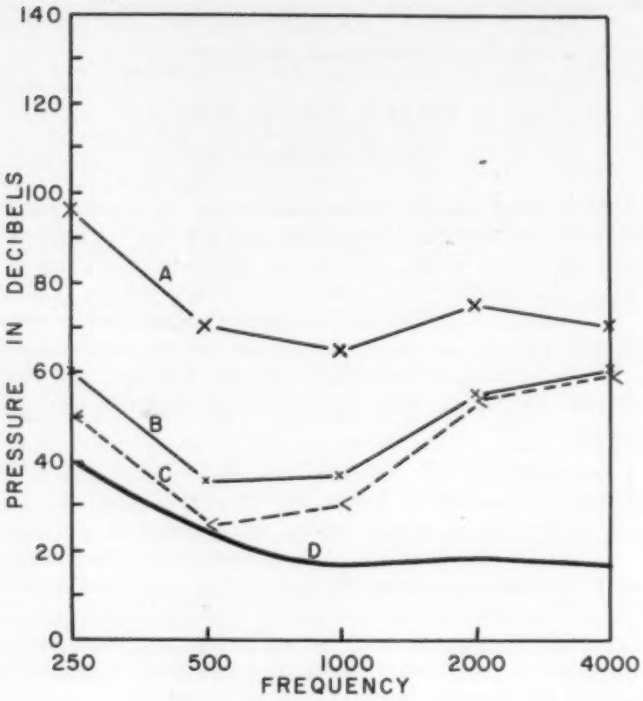

Fig. 1.

cornea lives almost by osmosis alone and is ectodermal in origin. The endothelium is not a true endothelium in the embryological sense.

Corneal grafts can be considered either lamellar or penetrating. The lamellar graft is one which will basically include only the epithelium, Bowman's membrane, and stroma. It is a split thickness corneal graft. A penetrating corneal graft is a full thickness graft, including the entire cornea's being lined with epithelium and "endothelium."

Corneal transplants are not new, being described by Dieffenbach[5] as an "audacious fantasy" over 100 years ago. The

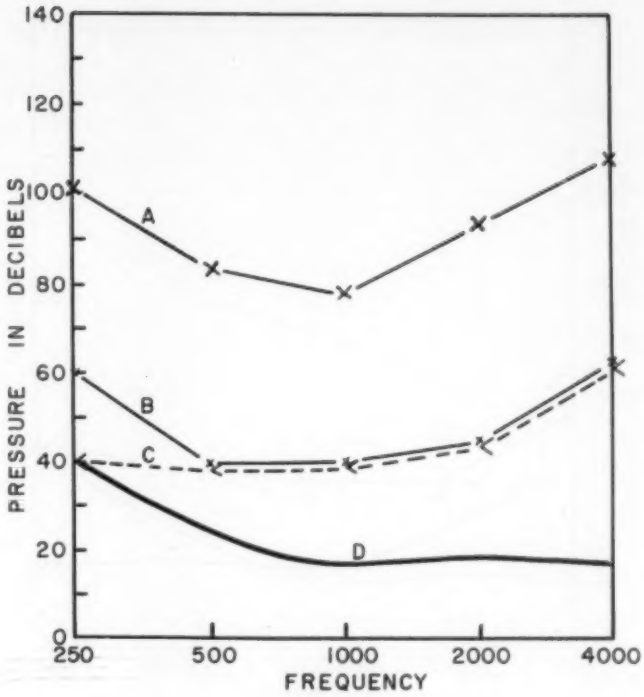

Fig. 2.

methods of obtaining, storing, and preserving corneas have been thoroughly studied and described.[6,7,8]

The procedures used to preserve corneas for use in keratoplasty are very specific, because ultimate transparency of the cornea is important. Since the utilization of the cornea in the ear is not concerned with opacification, corneas, not of value because of the ultimate opacity, can be used in ear surgery. Fresh penetrating or lamellar, and freeze-dry penetrating or lamellar corneas have been used in the ear.

The comparative greater thickness of the corneal graft aids in handling and positioning. Apparently, the thickness does

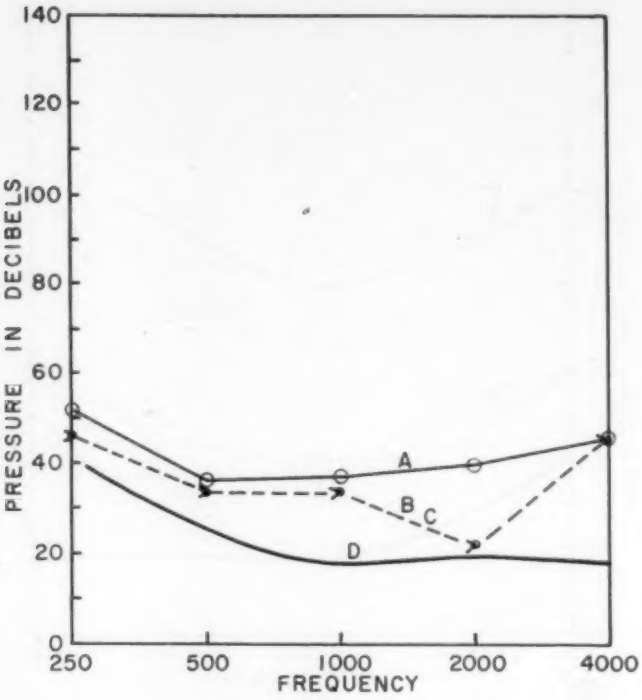

Fig. 3.

not interfere with the transmission of sound. Because of ease of handling, suturing of the graft to the remaining drum has been done using a special suture ligature and single strand raw silk worm gut. The immediate transparency in fresh grafts makes accurate positioning of the corneal graft easier.

Naturally, there are disadvantages to corneal grafts: first, human corneas are not readily available; second, the size of the cornea limits its use; third, antigen antibody reactions can occur, resulting in slough and sensitization. The first two can be eliminated by using heterografts.

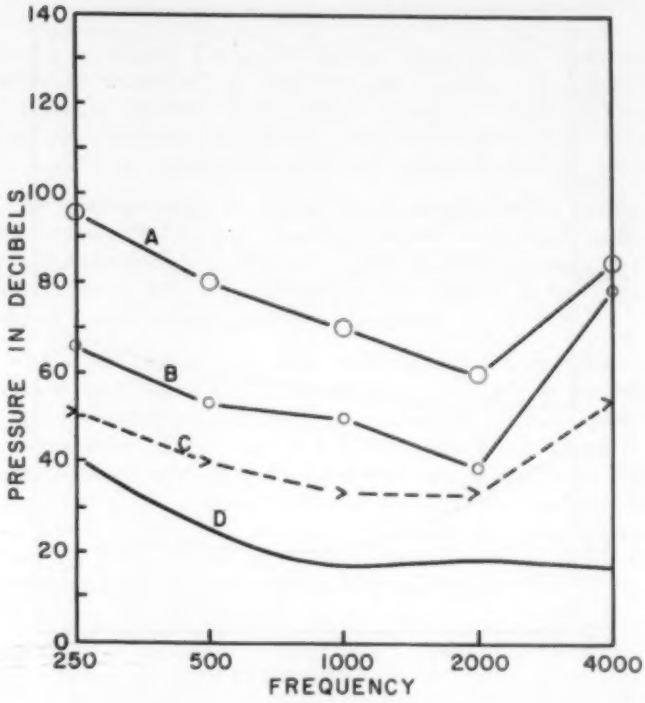

Fig. 4.

THE TECHNIQUES OF SURGERY.

It is not the purpose of this paper to discuss the surgical technique of tympanoplasties or stapedectomies. The technique of tympanoplasty and stapedial surgery was adequately discussed in the Workshop held in Chicago for Reconstructive Middle Ear Surgery in March, 1959.

This is a preliminary report of the use of corneal grafts in tympanoplasties and stapedectomies. Preparation of a donor site is done in the customary manner with special emphasis on hemostasis; otherwise, the advantage of transparency is lost. When a remaining viable part of the tympanic mem-

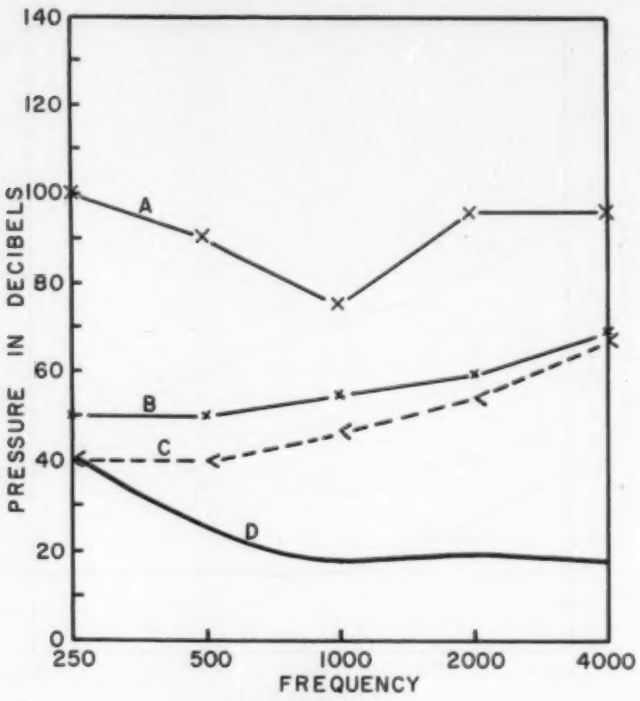

Fig. 5.

brane is left, the epithelium is removed from the drum, and a suitable sized cornea is sewed to the drum.

When exenteration of the mastoid cavity is necessary, the mastoid cavity is lined with a three-fourths thickness skin graft from a non-hairy portion of the body. The area tympanic membrane is replaced with a full thickness viable corneal graft, placed against the remaining movable ossicle or polyethylene tube.

Corneal homografts have been used in the place of vein grafts and gelfoam in stapedectomies. The cornea may be used either as a plug, similar to the gelfoam technique, or as a larger graft in the vein graft technique of Shea.

RESULTS.

Thus far in our limited number of cases, we have had uniformly good results. It has been interesting to note that in audiograms the low tones return sooner in stapedectomies than by using the gelfoam or vein graft techniques. The accompanying audiograms show the results in various cases.

The hearing pressure charts as illustrated utilize absolute pressures .0002 dyne per square centimeter as the base, rather than the customary corrective audiogram representing 0 as a normal level. The pressure grams are utilized as they show hearing levels rather than hearing losses.

In the pressure grams, Line A represents the preoperative hearing level; Line B, the postoperative hearing level; Line C, bone conduction; Line D, the average normal hearing pressure level. Figs. 1 and 2 are stapedectomies. Fig. 3 is a myringoplasty with a central perforation, having very little hearing loss. Fig. 4 represents a marginal perforation with destruction of the long process of the malleus. Fig. 5 represents the results of a Type III tympanoplasty in which the entire drum, incus, and malleus were destroyed.

CONCLUSION.

This is a preliminary report of the use of corneal homografts in the restoration of hearing in middle ear surgery. Corneal homografts appear to be a very satisfactory material for use in the middle ear because of the transparency and ease of handling. The results so far have been good.

A more exhaustive report of results and description of the individual techniques in the use of corneal grafts, both homografts and heterografts, will be presented in the future. A more detailed discussion of technique is being prepared and will accompany a future report.

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ANNUAL SPRING MEETING OF THE NEW YORK EYE AND EAR INFIRMARY ALUMNI.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 17-19, 1961.

Symposia will be offered on Otosclerosis and Benign Lesions of the Larynx. There will also be a Closed Circuit Television demonstration of surgical procedures. In addition there will be lectures on Plastic Surgery, Paranasal Sinuses and Carcinoma of the Head, Neck and Larynx.

For further information write Dr. John R. Finlay, Secretary, Alumni Association, 218 Second Avenue, New York 3, N. Y.

NEW METHOD OF TYMPANOPLASTY.

Preliminary Report.

RICHARD A. CHARLAND, M.D.,

Montreal, Canada.

Tremendous progress has been achieved in reconstructive surgery of the middle ear in the last few years, and unaided hearing has been restored to many patients suffering from cholesteatoma or chronic otitis media; however there are still some failures largely attributed to: *1.* the formation of scar tissue and adhesions to the stapes in the middle ear, and *2.* the necrosis and perforation of the skin graft intended to seal the middle ear.

Necrosis of the flap is due to poor blood supply to the free skin graft, and to postoperative infection.[1] A pedicled flap done according to established plastic surgery principles has a blood supply sufficient to prevent necrosis of the extreme end of the flap.[2] For this reason it should be no more than one and a half times longer than the width of the base, but in a highly vascularized area, when the blood vessels run parallel to the length of the flap, it can be made longer without danger of necrosis.

With these principles in mind I worked out a technique to use a pedicled post-auricular flap and to transpose it in the mastoid cavity and over the middle ear space. This flap may be used in Types II, III, and V.

I have used this technique most often in Type III and occasionally in Types II and V; therefore, I will describe my procedure as it is used in Type III.

STEP I. EXPOSURE OF THE MIDDLE EAR AND ATTIC.

An endaural incision is made, the middle ear, the attic, the

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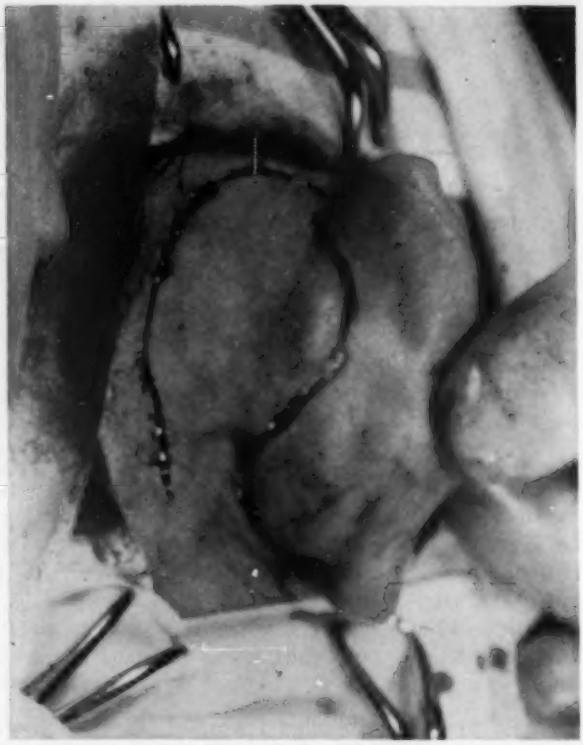

Fig. 1. The incision is made from the hair line to the posterior surface of the auricle through the skin and subcutaneous tissues, as illustrated.

ossicles and the antrum are exposed either by transcortical or subcortical approach.

STEP II. REMOVAL OF THE PATHOLOGY.

The pathology is removed from the middle ear, the antrum and the mastoid. When we are dealing with a cholesteatoma, it is removed completely under magnification, including the matrix. In cases of chronic infection, granulations, infected mucosa and infected bone are removed. Since the ossicular

Fig. 2. The flap is raised with blunt curved scissors from the post-auricular donor area.

chain is interrupted and the incus, the malleus and the tympanic membrane are badly damaged in these cases, they are removed; the remnants of the drum are also removed, but the stapes is preserved with as much mucous membrane as possible in the middle ear.[4]

STEP III. MOBILIZATION OF THE STAPES.

The stapes is mobilized by careful removal of adhesions, hypertrophied mucous membrane and cholesteatoma in this

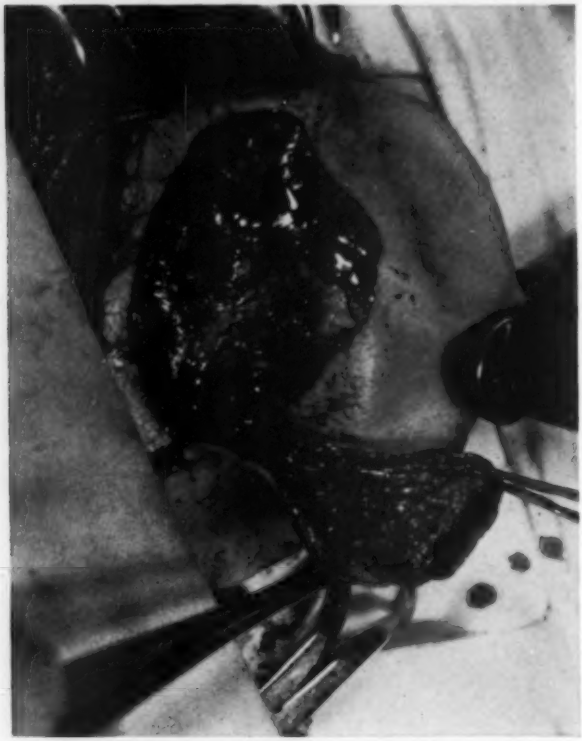

Fig. 3. The flap has been raised and its pedicle is shown near the mastoid tip.

area. The patency of the Eustachian tube is checked by means of a dull probe applied gently.

STEP IV. RAISING OF THE PEDICLED FLAP.

A pedicled skin flap is raised from behind the ear, the pedicle being at the mastoid tip and the free end near the upper attachment of the auricle as seen in Figs. 2 and 3. All the skin from the hair line to half way behind the pinna is raised. It is important not to include hair follicles that would

Fig. 4. A small incision is made through the periosteum near the mastoid tip to permit transposition of the flap to the middle ear through this incision.

otherwise grow in the mastoid cavity. The flap is made thin in the distal half and thicker in the proximal half.

STEP V. TRANSPOSITION OF THE FLAP.

The flap is transposed in the mastoid cavity. The soft tissues and periosteum are incised down to the mastoid process for a length of 2 cms., near the mastoid tip, this allows enough room for the transposition of the pedicled flap in the mastoid cavity and over the middle ear through the lower end of the

Fig. 5. The flap has been transposed to the mastoid cavity and is shown through the endaural incision.

post-auricular wound. The flap is placed in such a way as to seal the middle ear space and to be in close contact with the head of the stapes.[5] Packing is done with parresin mesh, and the endaural incision is closed.

STEP VI. CLOSURE OF THE DONOR AREA.

The edges of the post-auricular wound are undermined and are approximated with No. 000 silk mattress sutures, except for the lower third of the wound.

Fig. 6. The flap is being laid over the antrum and the middle ear.

Two or three months later the skin of the pedicle is incised, raised on the distal side, and the lower third of the wound is closed with No. 000 silk sutures.

In Type V, several months after the first operation has been done, and when the mastoid cavity is completely epithelized and there is no infection whatsoever, a tympanomeatal flap is incised and raised to expose the lateral semicircular canal as in fenestration surgery. A fenestration is made over the ampulla, the skin of the flap is thinned down and invaginated over the fenestra.[6]

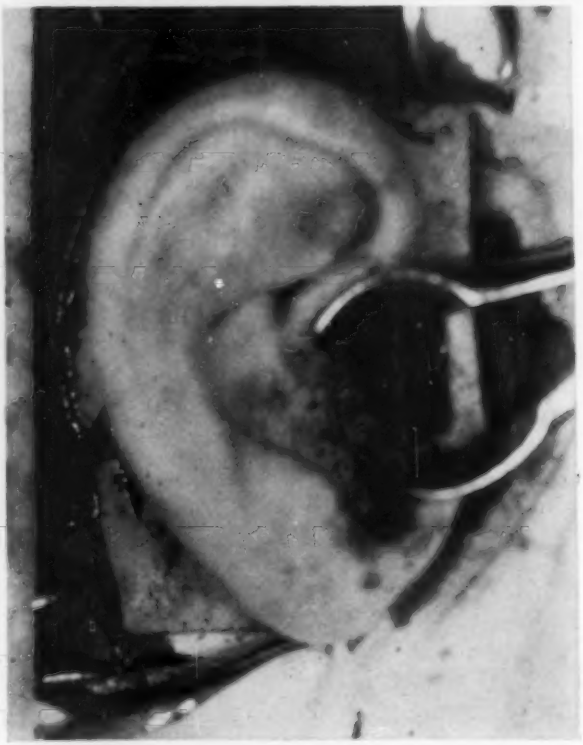

Fig. 7. The flap is shown sealing the middle ear and attic.

COMMENT.

The flap, if properly made, is long enough to reach and seal the middle ear space without tension on the pedicle[3]; in some instances there was even too much skin and some had to be cut off to allow the flap to fit properly. The transposition of the flap can be done without undue twisting of the pedicle which could interfere with the blood supply.[2]

The blood is supplied in this region by branches of the posterior auricular artery which run parallel to the length

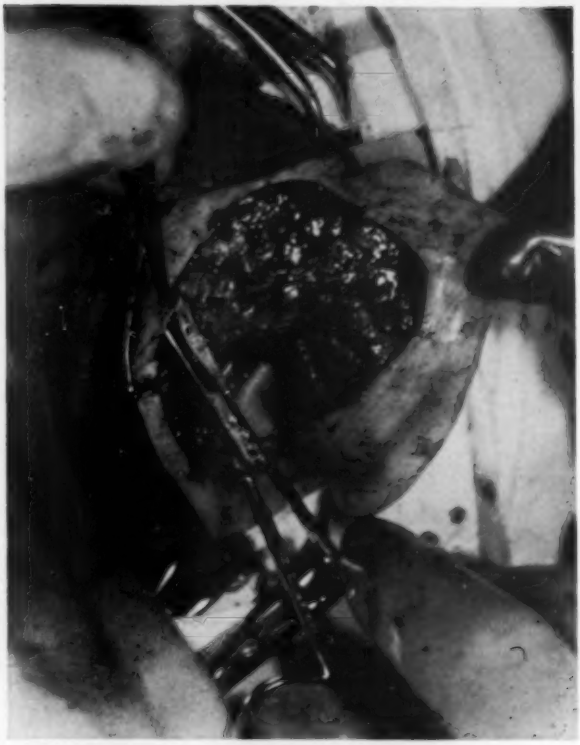

Fig. 8. Undermining is done with blunt scissors on each side of the wound for closure.

of the flap; this permits a length more than one-and-a-half the width of the base, without cyanosis and necrosis.

Inflation of the Eustachian tube is started ten days after the operation to prevent adhesions and to maintain an air space in the middle ear.

After closure of the post-auricular wound the auricle is somewhat closer to the skull than previously, but after a month or two the auricle has regained its original position. There is no deformity if the skin is raised no further than

Fig. 9. The donor area is closed with No. 000 silk mattress sutures.

half way from the post-auricular fold to the helix on the posterior surface of the auricle.

All this work is done extra-cartilaginous, the cartilage is not exposed or cut through at any time.

With this method, epithelization of the mastoid cavity is completed much earlier, and the danger of postoperative suppuration is much reduced on account of the rich blood supply that is brought in the mastoid cavity.[1]

I suggest the name "Middle Ear Reconstruction" to describe

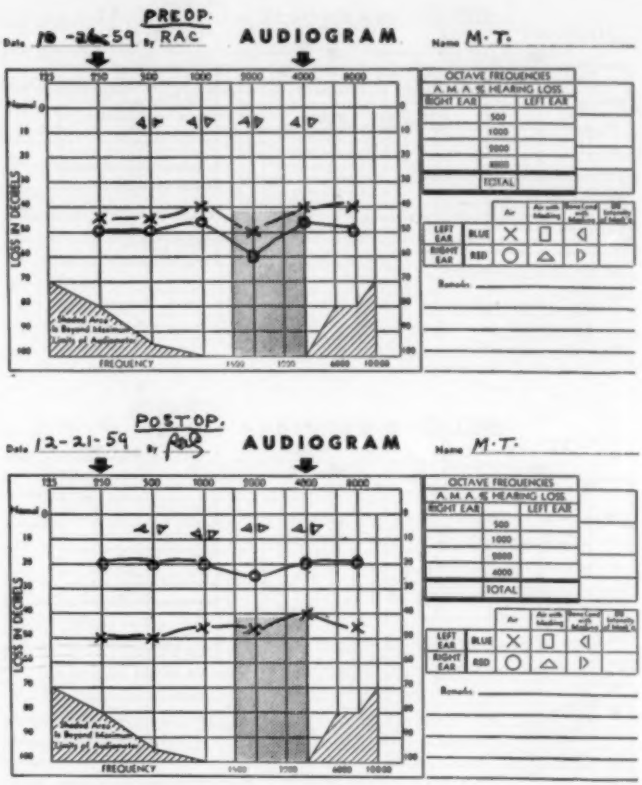

Fig. 10. Preoperative and postoperative audiograms in Type II tympanoplasty with post-auricular pedicle graft.

this procedure, whenever the pedicled post-auricular flap is used.

SUMMARY.

1. A pedicled post-auricular flap is recommended in tympanoplasty Type III and Type V, instead of a free graft.

2. This flap is intended to eliminate the postoperative necrosis and perforation of the graft that sometimes occur.

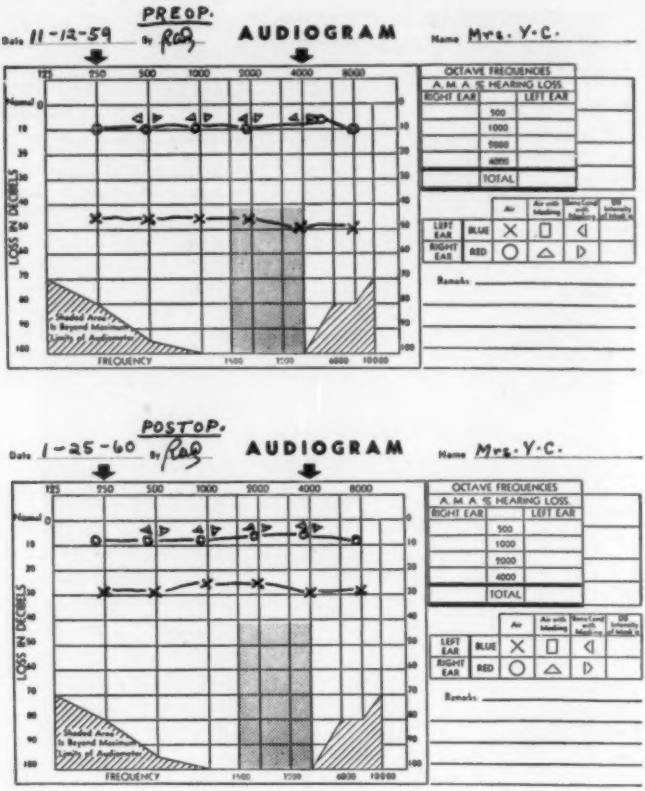

Fig. 11. Preoperative and postoperative audiograms in Type III tympanoplasty with post-auricular pedicle graft.

3. This flap carries with it a rich blood supply in the mastoid cavity, preventing, therefore, postoperative suppuration and promoting faster epithelization and healing.

4. There is no deformity of the auricle if the technique described is followed.

5. All the incisions are extra-cartilaginous to lower the risk of perichondritis and chondritis.

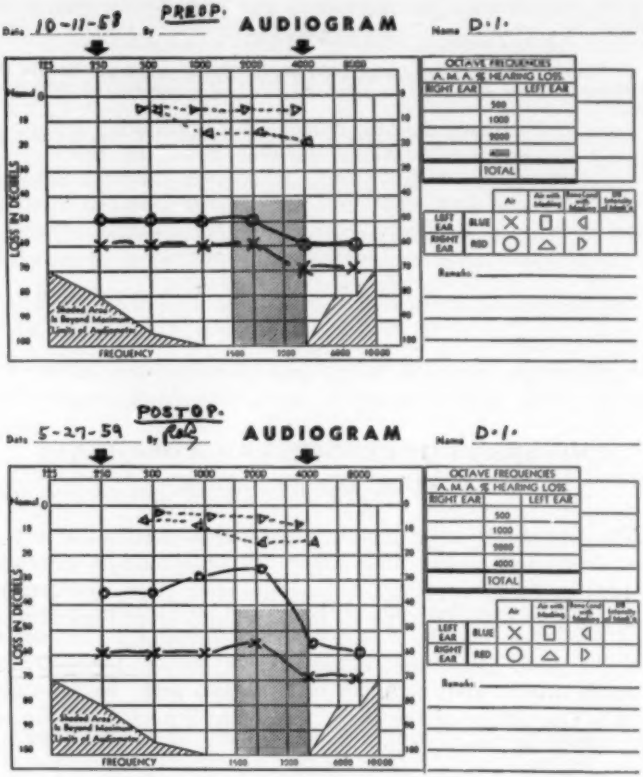

Fig. 12. Preoperative and postoperative audiograms in Type V tympanoplasty with post-auricular pedicle graft.

6. The name "Middle Ear Reconstruction" is suggested to describe tympanoplasty, whenever the pedicled post-auricular flap is used.

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Requests for these travel funds can be made by letter addressed to the Executive Secretary of the Selection Committee: Dr. Adolph L. Sahs, Department of Neurology, State University of Iowa, Iowa City, Ia.

The letter should include a brief summary of the applicant's educational, occupational, and research background, and should indicate his major field of interest. The letter should be signed by the applicant and countersigned by his administrative executive.

The deadline for the receipt of requests is March 1, 1961. The Selection Committee will inform applicants of its action by April 15, 1961.

Funds for this purpose have been made available through grants to the Selection Committee from The National Institute of Neurological Diseases and Blindness, Public Health Service; and the National Science Foundation.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

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Meeting: Palmer House, Chicago, Ill., October, 1961.

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Meeting: Lake Placid Club, Lake Placid, N. Y., May 23-24, 1961 (afternoons only).

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Annual Meeting: Chicago, Ill., October, 1961 (definite time and place to be announced later).

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Summer Meeting: June 28, 1961, Hotel Elysee, New York, N. Y.

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Meeting: Queen Elizabeth Hotel, June 15-17, 1961.

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Meeting: Oglethorpe Hotel, Wilmington Island, Savannah, Ga., March 2-4, 1961.

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Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

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Meeting: Third Thursday of November, January, February and April.

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Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 12-13, 1961.

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Meeting: Fourth Tuesday of each month from September through May, Aero Club, Portland, Ore.

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Meeting: Palmer House, Chicago, Ill., October, 1961.

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Executive Secretary: Dr. Chevalier L. Jackson, 3401 No. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.

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Place: Army and Navy Club, Washington, D. C.

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Meeting: Concurrent with the Academy of Ophthalmology and Otolaryngology, Chicago, Ill., October, 1961.

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Annual Meeting of the Section will be held in Dallas Tex., November, 1961.

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INDEX OF ADVERTISERS

	Page
American Academy of Ophthalmology and Otolaryngology Registry	19
American Cystoscope Makers, Inc.	2
American Laryngological Association	19
Back Issues Wanted	7
Beltone Hearing Aid Company	18
Books Received	19
Burroughs Wellcome & Co., Inc.	8-11-20
Central Institute for the Deaf	21
Ciba	13
Doho Chemical Corp.	15
Eaton Laboratories	12
E.E.N.T. Practice for Sale	19
S. E. Massengill Co.	3-4
V. Mueller & Co.	10
Nichols Nasal Syphon, Inc.	14
Notice to Subscribers	Inside Back Cover
Otarion Listener Corp.	9
G. P. Pilling & Son Co.	6
Radium Chemical Company, Inc.	16
Roche Laboratories	Back Cover
Storz Instrument Co.	5
Surgical Mechanical Research	17

RS

Page

gy
.....19
..... 2
n....19
..... 7
.....18
.....19
-11-20
.....21
.....13
.....15
.....12
.....19
...3-4
.....10
.....14
over
..... 9
..... 6
....16
over
..... 5
....17

V
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INDEX ISSUE

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INDEX ISSUE

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CONTENTS

HISTOPATHOLOGIC OBSERVATIONS IN SO-CALLED CONGENITAL FIXATION OF THE STAPEDIAL FOOTPLATE. John R. Lindsay, M.D., Chicago, Ill., Sam H. Sanders, M.D., Memphis, Tenn., and George T. Nager, M.D., Baltimore, Md. - - - - - - - - - - 1587

STAPES SURGERY. Juan M. Tato, M.D., Buenos Aires - - - - - 1603

VENTRICULAR DYSPHONIA: NEW INTERPRETATION OF AN OLD OBSERVATION. Godfrey E. Arnold, M.D., New York City, N. Y., and Samuel Pinto, M.D., Mexico City, D. F. - - - - - - - - 1608

THE PSYCHOLOGICAL MANAGEMENT OF THE SURGICAL PATIENT: VOODOO, MESMER AND ALEXANDER POPE. Floy Jack Moore, M.D., Jackson, Miss. - - - - - - - - - - - - - - - - - - 1628

MULTIPLE PRIMARY MALIGNANT TUMORS. Phillip A. Dibble, Captain, MC, USA, and Robert G. Chambers, M.D., Washington, D. C. - 1647

A NEW TECHNIQUE FOR MYRINGOPLASTIC PROCEDURES. Dieter Wendling, D.D.S., M.D., Royal Oak, Mich. - - - - - - - - 1658

ETHER ANESTHESIA FOR TONSILLECTOMY STUDIES IN RESPIRATORY ADEQUACY. Thomas D. Graff, M.D.; Leroy C. Harris, M.D.; Thomas D. Michael, M.D.; Otto C. Phillips, M.D.; Alfred T. Nelson, M.D., and Edward T. Fox, M.D. - - - - - - - - - 1660

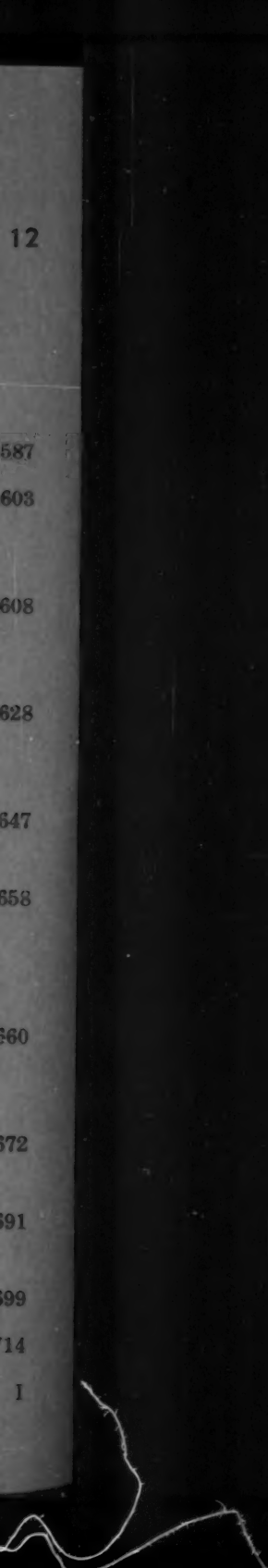
CURRENT UTILIZATION OF TRACHEOTOMY AS A THERAPEUTIC MEASURE. A REVIEW OF THE LITERATURE AND AN ANALYSIS OF 526 CASES. Joseph P. Atkins, M.D., Philadelphia, Pa. - - - - - 1672

CORNEAL GRAFTS IN MIDDLE EAR SURGERY. A PRELIMINARY REPORT. Frank S. Forman, M.D., Colorado Springs, Colo. - - - - - - 1691

NEW METHOD OF TYMPANOPLASTY. PRELIMINARY REPORT. Richard A. Charland, M.D., Montreal, Canada - - - - - - - - - - 1699

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES - - - - - - - - - 1714

INDEX FOR THE YEAR 1960—SUBJECTS AND AUTHORS - - - - - - I



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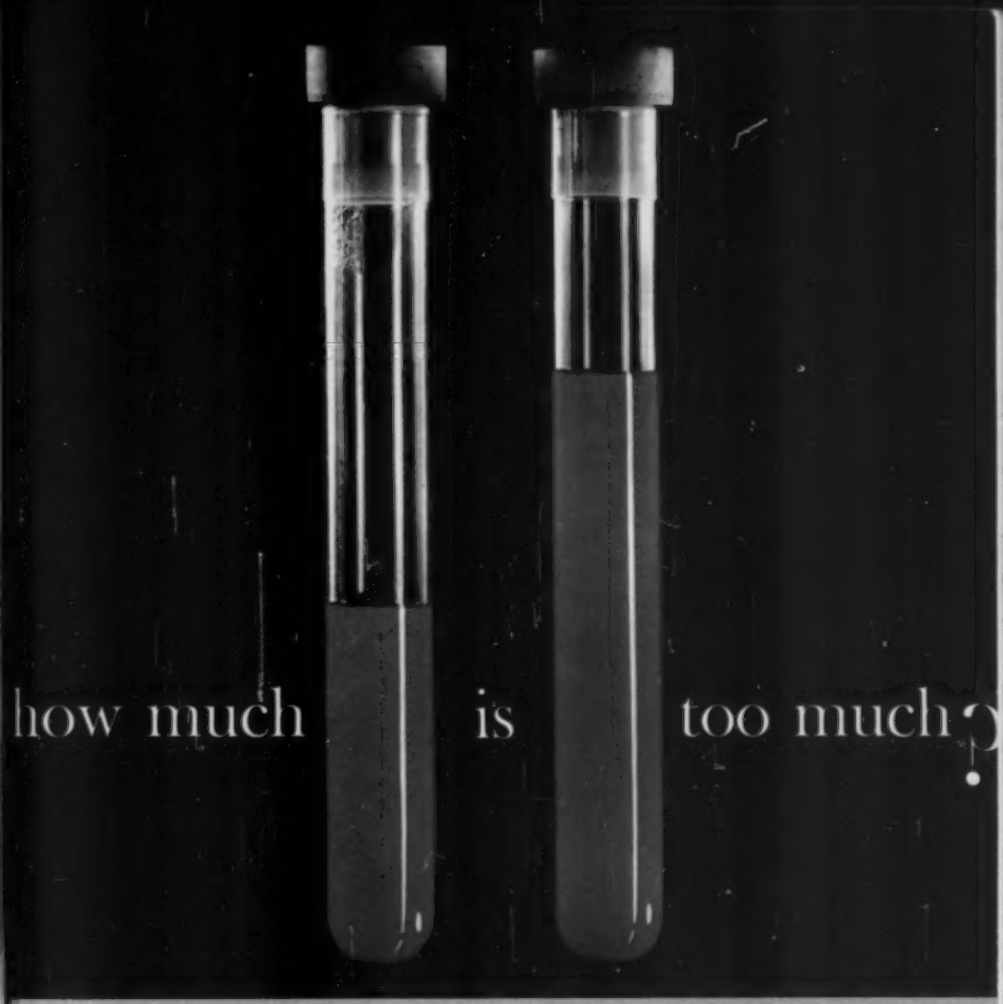

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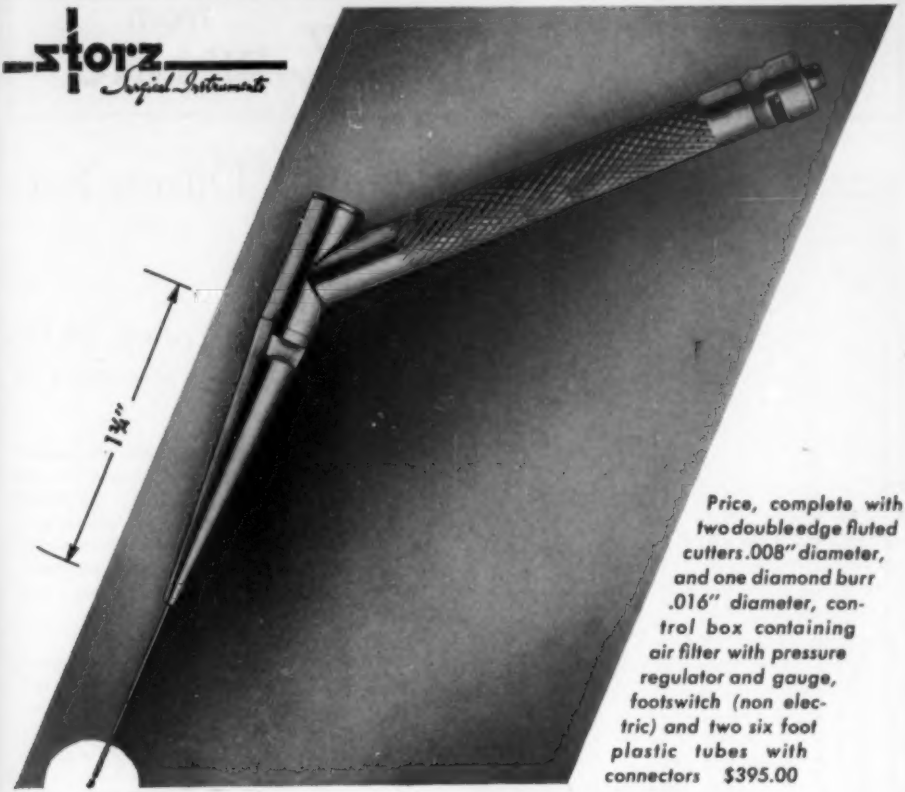

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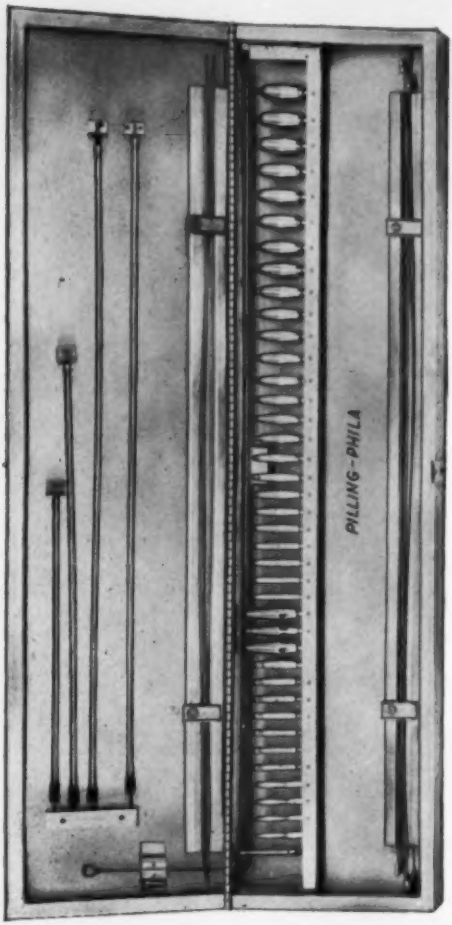

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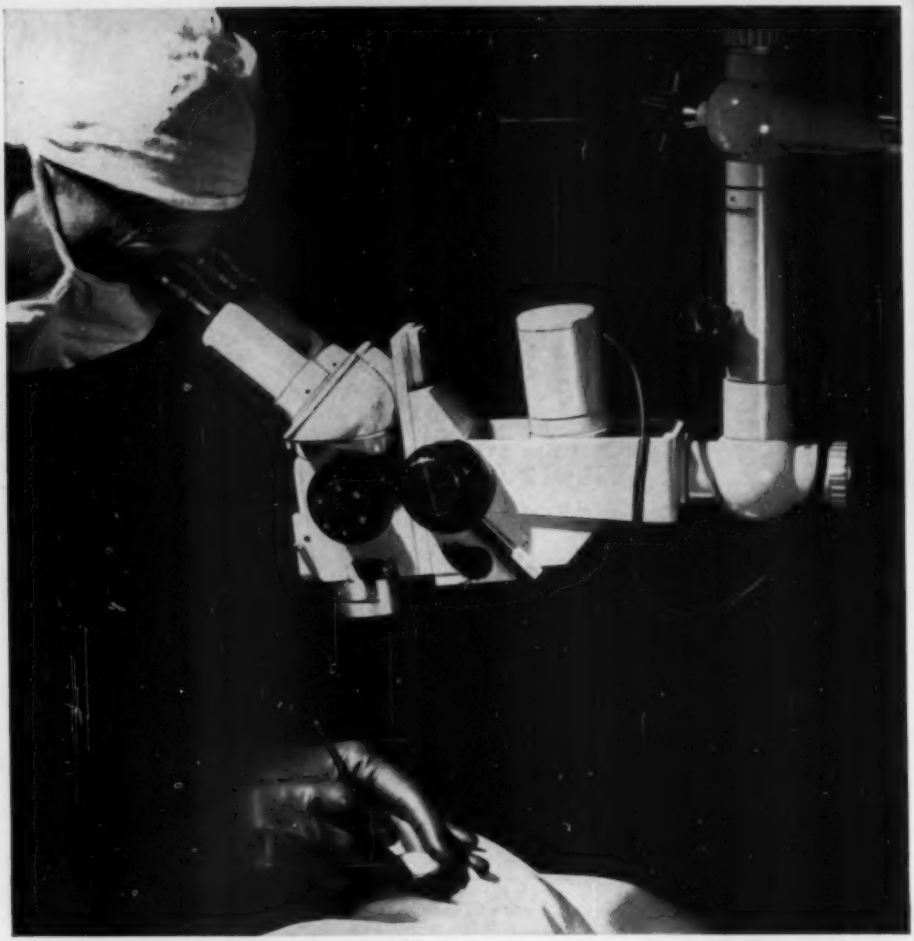

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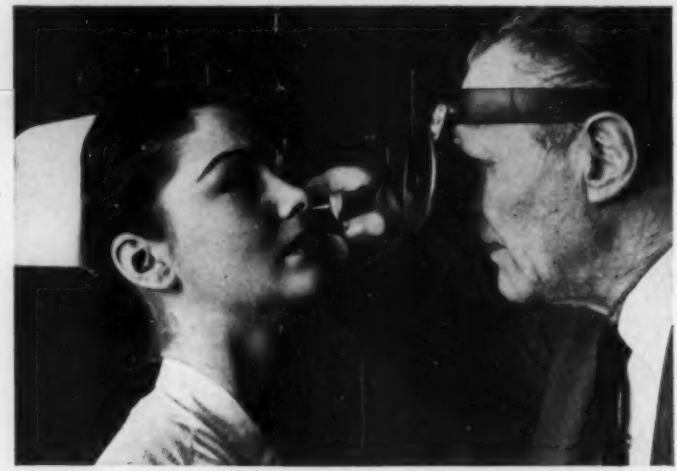

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